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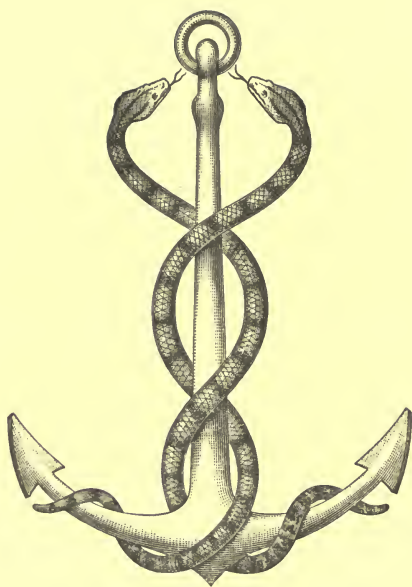




*Pentland's Medical Series.*

VOLUME SIXTH.

DISEASES OF THE HEART AND  
AORTA.



NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

MC  
G.

# DISEASES

OF THE

# HEART AND AORTA

BY

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TO

GEORGE WILLIAM BALFOUR,

M.D. ST. AND., LL.D. ST. AND., AND EDIN., F.R.C.P.ED., F.R.S.E.

CONSULTING PHYSICIAN TO THE ROYAL INFIRMARY OF EDINBURGH,

IN ACKNOWLEDGMENT OF HIS IMPORTANT SERVICES TO

THE STUDY OF DISEASES OF THE HEART

AS WELL AS IN TOKEN OF

RESPECT AND ESTEEM.



## PREFACE.

---

IN the preparation of this work it has been my object to give a faithful reflection of the state of medical science and art at the close of the present century. While attempting to reach an adequate explanation of every subject attended by difficulty, my chief care has been to keep before me throughout the aim of practical utility. With this end in view the different aspects of disease have been illustrated by clinical examples, and the various details of treatment have likewise been exhibited in typical cases. Whenever possible these have been taken from the results of hospital experience, and only when such instances were not obtainable have private case-books been utilised.

References to previous authors have been given with a freedom which may possibly be considered profuse. We are all, however, under a debt of gratitude to those into whose labours we have entered, and it is accordingly our bounden duty to render suitable acknowledgment.

Seeing that my work has been aided and lightened by the kindness of friends too numerous to mention, it may seem almost invidious to refer specially to any ; but certain of them have laid me under a weight of obligation which can never be adequately repaid, although gratefully recognised. To Sir

Thomas Grainger Stewart my deep acknowledgments are cordially rendered for the more than generous manner in which he at all times encouraged me to utilise the opportunities afforded by his wards during the years we were associated in the Royal Infirmary. By Professor Cunningham, Dr. Noël Paton, Professor Muir, and Professor Stockman the greatest assistance has been given me in writing those portions of the volume which deal with the special departments they severally cultivate, and my warm thanks are now returned for their unwearied kindness.

In the verification of the references invaluable services have been rendered by Mr. J. Matheson Shaw and Mr. J. Y. W. MacAlister, Librarians to the Royal College of Physicians of Edinburgh, and to the Royal Medical and Chirurgical Society of London respectively; to these gentlemen my obligations are heartily acknowledged for their unvarying courtesy.

The illustrations, with the exception of a few taken from previous works of my own, have been entirely obtained for this volume. Those which deal with objects seen with the naked eye are mostly due to Mr. Hume Paterson, of the Laboratory of the Royal College of Physicians of Edinburgh, while the reproductions of microscopic appearances have all been drawn by Mr. Richard Muir, of the Pathological Laboratory of the University of Edinburgh, to both of whom my thanks are cordially given.

17 ALVA STREET, EDINBURGH,  
26th September 1898.

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DISEASES OF THE HEART AND  
AORTA.



# DISEASES OF THE HEART AND AORTA.

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## CHAPTER I.

### MORPHOLOGICAL.

#### INTRODUCTORY.

THE aim of the circulation is to render possible the continuous interchange between the blood and the tissues through which it flows. If this interchange were a constant quantity, the maintenance of the circulation would be a simple physical problem, and a purely mechanical apparatus would serve all its purposes. The metabolic processes, however, are subject to great fluctuations. There are not only changes occurring through the entrance of new materials from the alimentary viscera, but there are also modifications produced by intrinsic activities, as well as alterations caused by external agencies. These and many other analogous influences render some means of adapting the circulation to the wants of the tissues an essential and necessary condition for the maintenance of life. And, if the variations taking place under conditions which may be considered within the limits of health, demand some means of adjustment, such a possibility of adaptation is much more required to provide for the alterations effected by disease, whether occurring in the form of changes in the composition of the blood, or of modifications in the structure of the tissues through which it flows. There are thus some purely mechanical questions connected with the provision for

a circulating medium, and there are also some complex vital problems relating to the adjustment of that nutritive and depurative medium in varying conditions.

In order to have a thorough understanding of the circulation it is necessary to study it from every point of view, and the difficulty in such a work as this is to decide what it is needful to include, and what it may be possible to omit. All facts which can elucidate the subject are of use, but some of them are of much greater importance than others; to present the subject, therefore, in a proper perspective requires the exercise of considerable selective judgment.

It may seem needless to enter upon a discussion of embryological facts in a work written, as this is, with a view to practical ends. In recent years, however, every department of medicine has largely gained by a study of the phases of development, and that dealing with the circulation at least as much as any other. The short sketch of the circulation during embryonic life, with which this work begins, is restricted to the consideration of such points as are necessary to bring into relief the main facts of development, and to provide the data from which important general conclusions may be drawn.

How far the introduction of anatomical and physiological considerations is necessary or expedient in such a volume as this must always be a question difficult to decide. Many facts with reference to these aspects of the circulatory organs must be regarded as familiar to every one, and it would be simply a waste of time to recall them. But before entering upon the study of the heart in diseased conditions it is clearly necessary to have a definite conception of medical anatomy and physiology. Such a conception renders the description of the special affections of the heart and great vessels a matter to be more easily accomplished, and at the same time facilitates a complete grasp of the whole subject, more especially if the facts of general pathology, as applied to the circulation, receive preliminary consideration.

A systematic analysis of the general symptomatology, and of the principles of therapeutics, with regard to the circulation, will also be found to facilitate the comprehension of many

details belonging to the special diseases to be afterwards discussed.

Holding such views, it has therefore seemed to me essential to give a somewhat full preliminary statement of many matters of a general character, in order to pave the way for their systematic consideration in the various special departments into which they naturally fall.

### EMBRYOLOGICAL CONSIDERATIONS.

On account of difficulties inseparable from the subject, there are many gaps in our knowledge of the development of the human embryo. As the general history of the process, however, is closely similar to that seen in other mammals, it is possible, notwithstanding certain differences in detail, to supply many of these deficiencies. In the following short account of the development of the circulatory system, the facts are taken as far as is possible from observations on the human embryo; many points, nevertheless, are elucidated by reference to what has been described in other mammals, and even in animals belonging to lower vertebrate classes.

**THE HEART AND GREAT VESSELS.**—The vascular system makes its first appearance in the form of two simple lateral tubes in the anterior part of the embryo, occupying spaces continuous with the pleuro-peritoneum; they are composed of two layers, apparently developed respectively from the mesoblast and hypoblast, and are continuous in front with the cephalic mesoblast, while connected behind with the omphalo-meseraic or vitelline veins. These tubes are developed in the lateral part of the embryo, which, on the folding downwards of the body walls, becomes the ventral wall of the pharynx; and when this wall is completed they meet in the middle line, and become fused so as to form a single channel. This tube remains in connection with the vitelline veins behind, and bifurcates in front, giving rise to the primitive aortæ.

At this stage in the development of the vascular apparatus, the single median tube begins to pulsate; a fact of far-reaching importance, which will be described more fully in the sequel.

The central vascular tube, at a slightly later stage, becomes curved by bending over to the right; and at the same time its posterior extremity, connected, as already seen, with the venous channels behind, assumes a position dorsal to the other parts. At this period of development, superficial constrictions appear upon the tube, dividing it into the venous sinus, the auricle, the ventricle, and the aortic bulb. While these changes are going on, muscular fibres are for the first time seen in the substance of the vascular apparatus. This is another important fact, for the appearance of pulsation before the development of muscular tissue raises some interesting problems.

The venous sinus receives the posterior veins already mentioned, and the primitive veins from the cephalic region. It at first communicates freely with the auricle, but afterwards is guarded by venous valves. The common auricle continues to develop behind the curved ventricular portion of the tube, and in time the right venous valve forms the Eustachian and Thebesian valves, while the left venous valve disappears. The common ventricle, or curved ventral portion of the tube, receives the blood from the common auricle at its left end, and terminates at its right end in the aortic bulb. It contains in its early stages a fine spongy substance. A slight contraction on its outer surface marks the future separation into right and left ventricles, and a septum, composed of muscular tissue, begins to grow upwards and forwards from below and behind. This is completed at a later stage by the development of a fibrous septum, which grows downwards from above and before to join the partial muscular partition. At the same time the spongy tissue in the cavity of the ventricular portion of the apparatus begins to be gathered together in distinct masses—the *musculi papillares* and *columnæ carneæ* of the fully developed heart.

The common auricle is gradually raised upwards and forwards, so that the opening into the ventricular portion assumes a position over the inter-ventricular septum, which grows up, and, with the assistance of flaps, to be described below—the future auriculo-ventricular valves—divides the orifice into two divisions. A septum is developed in the common auricle from



above and behind, downwards and forwards, in which the foramen ovale is formed. Before this inter-auricular septum is produced, the pulmonary vein is developed.

The foramen ovale is freely open until the fourth month, but about that period the valvular apparatus guarding the opening is developed, and in the last three months of foetal life the blood can only pass from the right to the left auricle.

While these changes are proceeding, the aggregation of the spongy tissue of the ventricles into distinct masses goes on, and by this means the columnæ carneæ, as well as the muscoli papillares with their chordæ tendineæ, are formed; the latter becoming attached to the flaps developed at the auriculo-ventricular orifices. The inter-ventricular septum grows up into the aortic bulb and separates it into two divisions, *i.e.* aorta and pulmonary artery, each communicating with its respective ventricle. Folds of the lining membrane grow up at the junction of the bulb and the vessels, and by their division into segments form the semilunar valves.

The auriculo-ventricular valves appear, from the researches of the younger His, to be composed, in birds and mammals, of five layers, derived from : (1) the endocardium of the auricle; (2) the muscular wall of the auricle; (3) the pericardium, which breaks through the muscular wall in the auriculo-ventricular region, and forms the greatest part of the valve in the adult; (4) the ventricular muscle—a layer specially related to the papillary muscles, when they are developed; and (5) the endocardium of the ventricle. The two muscular layers become gradually altered, that from the ventricle passing into fibrous tissue attached to the chordæ tendineæ, and that from the auricle forming the muscular tissue of the auriculo-ventricular valves discovered by Kürschner and more fully described by Joseph and Darier.

The development of the great arterial trunks must receive some attention. From the aortic bulb two arterial arches spring, each running forwards, outwards, and backwards, behind the primitive heart, to form the primitive aorta of its own side. The two primitive aortæ unite at an early period in the middle line about the dorsal region. To these arterial arches four other lateral pairs are in succession added, forming

five on each side. Of these arches the first and second become the external carotid artery and its branches ; the third forms the internal carotid artery. The fourth, on the right side, becomes the subclavian artery, and on the left side it forms the arch of the aorta. The fifth, on the right side, develops as the right pulmonary artery, and its distal portion vanishes ; on the left side it forms the left pulmonary artery, and the portion beyond remains during foetal life as the ductus arteriosus. The right descending primitive aorta entirely disappears in its anterior part ; the left remains as the permanent descending aorta, and joins the posterior part of the right primitive aorta behind, to form the posterior portion of the permanent descending aorta.

Haller pointed out that the heart in the embryo of the fowl begins to pulsate before any other structure shows a trace of irritability, and Bischoff attributed this to an inherent tendency in the heart muscle ; inasmuch, however, as the pulsation begins before the appearance of any muscle cells—a fact first noted by Eckhard—we are driven to conclude that rhythmic pulsation may be independent of muscular structure. Since the heart movements begin before the evolution of any nervous elements, as was shown by Preyer, it is obvious that they must be due to some, as yet unknown, indwelling property of the embryonic heart tissue.

From the researches of the younger His, it appears that in the embryo of the fowl the heart commences to pulsate about thirty-six hours after the beginning of incubation. The pulsation is at first irregular, but after the first two days have passed it becomes regular ; its rate varies with the external temperature.

About the fourth or fifth day the structure of the heart wall is considerably altered. Hitherto the cells of which it is composed are circular, but about this period they become elongated and begin to form the trabeculae on the inner aspect of the walls. At the same time the pulsation changes its character. Hitherto a simple peristaltic wave, passing uniformly over the entire cardiac structures, it now begins with a pulsation of the venous sinus, which, after a slight pause, passes over the auricles ; then, succeeding a longer pause,



ensues the ventricular contraction, followed immediately by the pulsation of the aortic bulb. At this time no nervous structures have made their appearance, and it is not until the sixth day that ganglionic cells are found.

From the researches of Fano it is quite clear that from the outset the individual parts of the embryonic heart possess different characteristics; they react differently, for example, to stimuli, and their behaviour under the influence of cardiac poisons is not the same.

The development of the cardiac ganglia in the embryonic heart does not, according to His, produce any noteworthy change in the action of the heart, the phenomena observed on the seventh day of the embryo of the fowl being in all respects similar to those seen on the fifth. These observations, originally made on the fowl, have been verified by the same observer in fishes, frogs, and rabbits. It is worthy of remark that Pflüger observed pulsation of the heart of a human embryo of three weeks, and it is well known that the earliest ganglia appear in it at the end of the fourth week. It is impossible, after considering these observations, to avoid the conclusion that the heart is endowed with a tendency to rhythmic pulsation, altogether independent of any special nervous stimulus.

During the later stages of foetal life the circulation has certain peculiarities of arrangement, which disappear soon after birth. In the first half of intra-uterine life the foramen ovale, as has been noted above, is freely open, but during the later months of foetal existence, in consequence of the development of the valve of the foramen, it is only open for the passage of blood from the right to the left auricle. At this later period also the Eustachian valve has come into existence, and directs the current of blood, reaching the right auricle by the inferior vena cava, towards the foramen ovale. By means of the ductus arteriosus, whose origin has been described, there is free communication between the aorta and pulmonary artery; this does not cease until the obliteration of the ductus, usually at the end of the first week after birth. This free opening between the two great vessels must of necessity tend to equalise the blood pressure in them.

As is well known, the thickness of the walls of the two

ventricles is almost equal during foetal life, a fact which seems obviously due to the nearly equal pressure caused by the communication between the two sides of the system, as was, indeed, suggested by Harvey. In order to ascertain the relation borne by each ventricular wall at different stages of growth, the subject has within recent times been investigated by Lockhart Gillespie and myself. The method followed was to make a series of sections through the ventricular portion of the heart of the foetus at right angles to its axis, and measure the thickness of the walls half-way between the auriculo-ventricular groove and the apex.

The following is a summary of the results :—

	Wall of Right Ventricle.	Wall of Left Ventricle.
1. Fœtus, 3½ months . . . . .	1·5 mm.	1 mm.
2. Fœtus, 4 months . . . . .	1·5 „	0·5 „
3. Fœtus, 6 months . . . . .	4 „	3 „
4. Fœtus, full term, never breathed . . . . .	4 „	3 „
5. Fœtus, full term, never breathed . . . . .	5 „	5 „
6. Fœtus, full term, lived 24 hours . . . . .	3 „	4 „
7. Female infant, 11 months . . . . .	2 „	5 „
8. Male infant, 22 months . . . . .	2 „	8 „

These figures show that the left ventricle progressively increases in thickness from the third month, while the right ventricle increases up to the hour of birth, and then rapidly diminishes, the thickness of its wall nearly two years afterwards being actually smaller than in the sixth month of foetal life. From these facts it is clear that the thickness of the walls of the cardiac chambers is proportional to the work which each has to do. They are in every respect supported by observations made by us on the foetal heart of the horse, cow, sheep, pig, dog, and cat.

The consideration of these points in development is by no means a matter only of scientific interest; on the contrary, the facts which have been briefly described in the preceding pages render it more easy to show how different malformations are possible through arrested or perverted development. They point, moreover, to certain important truths. The pulsation of the primitive vascular tube, for instance, before the growth of any muscular fibres in its sub-

stance, appears to be evidence in favour of the existence of an inherent tendency to rhythmic propulsive movements in the vascular mechanism. And, as during foetal life the blood pressure must be nearly the same on both sides of the heart, it is undoubtedly a circumstance of real importance that up to the time of birth the thickness of the walls of the two ventricles is almost equal on the two sides. This fact may be taken to prove that the amount of strength developed is strictly proportional to the work that has to be done. Such results lead to far-reaching conclusions in regard to the possibility of the heart adapting itself to widely different morbid conditions.

**THE BLOOD VESSELS.**—The blood vessels take their origin in the mesoblast and hypoblast. They appear first as irregular spaces and canals, formed by the separation from one another of the mesoblast cells. The cells surrounding these spaces undergo modification in character and arrangement, and become altered so as to form blood vessels. But the network which is produced becomes connected with the hypoblast, from which are derived the capillaries, as well as the endothelial lining of the larger blood vessels, around which the muscular and connective tissue grows from mesoblast structures. The primitive blood corpuscles appear to have a double origin; some are cells enclosed within the lacunæ, others are produced by budding from the walls of the vessels.

**THE COURSE OF THE BLOOD.**—The course of the foetal circulation differs from that of the child after birth on account of the special conditions imposed by intra-uterine existence—the presence of the placental arrangement for purifying the blood, and the absence of any pulmonary functions. The extension of the hypogastric as umbilical arteries, ending in the placental capillaries, and the presence of the single umbilical vein, which represents the left allantoic vein—the right having disappeared—terminating, by means of the ductus venosus, in the inferior vena cava, form the mechanism which provides for the placental circulation. The absence of any respiratory function in the lungs necessarily removes almost entirely the need for a free supply of blood by the pulmonary circuit, and apparently the pulmonary artery only conveys enough of blood to its branches to keep them

open. In order to carry out this distribution two structural provisions are present in the foetus. By means of the foramen ovale, much of the blood returned by the inferior vena cava flows from the right into the left auricle, and through the ductus arteriosus a large proportion of the blood sent into the pulmonary artery finds its way into the aorta.

At birth, the commencement of the respiratory functions of the lungs and the arrest of the placental circulation produce changes in the course of the blood, attended by structural alterations in the heart and vessels. The blood begins to flow freely through the lungs, and the hypogastric vessels are left empty. The ductus venosus becomes obliterated as well as the umbilical vein; the foramen ovale becomes closed; and the ductus arteriosus together with the hypogastric arteries shrivel up. All these changes are effected within eight days from the date of birth, and henceforth the course of the blood is as in adult life.

#### ANATOMICAL CONSIDERATIONS.

The facts regarding the anatomy of the circulatory apparatus may fitly begin with the heart and its relations; reference to the peripheral blood and lymph vessels being made afterwards.

PERICARDIUM.—The pericardium may most conveniently be considered from the point of view of its two-fold arrangement, comprising as it does the parietal or outer, and the visceral or inner portion, often termed the epicardium.

The parietal pericardium consists of two layers, the external or fibrous and the internal or serous. Although closely applied to the structures which it covers and protects, it is nevertheless extremely extensible, no doubt on account of the large proportion of yellow elastic fibres which it comprises. Resting below on the central tendon, with which it is intimately connected, and in front on the anterior muscular portion of the diaphragm, the fibrous pericardium extends upwards in an irregularly conical form to a point about two inches above the origin of the aorta. It is attached in front by two fibrous bands, often called the superior and inferior sterno-pericardial



ligaments, to the body of the sternum and to the xiphoid cartilage. Posteriorly it is attached to the vertebral column by the fibrous tissue of the posterior mediastinum. At its upper extremity it has four openings—for the aorta, the two divisions of the pulmonary artery, and the superior vena cava, respectively. Posteriorly there are also four apertures through which the four pulmonary veins pass, and in foetal life there is also an opening for the ductus venosus. The fibrous pericardium is everted at these openings, and is intimately united to the outer coats of the vessels. As the vessels have reflections of the cervical fascia, the pericardium is indirectly attached to the clavicle and upper ribs. The inferior surface of the fibrous pericardium presents an opening for the passage of the inferior vena cava, but here the membrane is not everted in consequence of its relation to the diaphragm, and it does not become so closely incorporated with the wall of this vessel as with the outer coat of the others which pass through it. It is not to be forgotten that the fibrous pericardium is covered on its outer surface by the parietal layer of the pleura throughout great part of its extent, only an elongated triangular portion in front, and an irregular area behind, in connection with the great vessels, being uncovered by the pleura.

The serous lines the fibrous layer of the pericardium throughout its whole extent, excepting a small area posteriorly where they are separated by some of the great venous trunks. The fibrous pericardium may therefore be regarded as invested on both aspects with serous membrane to a great extent, so that it is often termed the pleuro-pericardium. The serous layer is reflected from the fibrous envelope upon the aorta and pulmonary artery to form the visceral pericardium. These vessels have a common serous covering which extends over them for about two inches from their origin. Passing from the root of the aorta and pulmonary artery to the auricles, the visceral pericardium envelops the entire external surface of the heart, the anterior aspect of the superior vena cava for about an inch, and the anterior, inferior, and posterior surfaces of the pulmonary veins to a variable extent.

EXTERIOR OF THE HEART.—The form of the heart changes greatly, not only in its varying phases of activity, but also in the

different positions it may be placed in. Many discrepancies in the descriptions which have been given of it are probably therefore dependent on its having been described while in different stages of contraction or relaxation, or in diverse positions. The best general idea of the organ as a whole is undoubtedly obtained by studying a specimen which has been fully injected; this, however, although presenting the fully distended condition of every part, is misleading, inasmuch as it produces a state of matters which does not occur during life, since the auricles and ventricles are never at the same time fully distended.

The heart presents an irregularly and obliquely conical form, flattened in such a way as to show three surfaces, rather indefinitely bounded by five borders.

The anterior surface, which is convex and generally regarded as somewhat triangular in outline, is in reality of an irregularly oblique rhombic shape. It is formed by the right auricle with its appendix, the right ventricle, and the left ventricle, as well as by the roots of the aorta and pulmonary artery, and by the tip of the appendix of the left auricle. Its component parts are separated from one another by the auriculo-ventricular, and the inter-ventricular sulci, in which lie the coronary arteries, with the cardiac veins and the coronary plexuses.

The posterior surface, which is quadrangular and convex, is formed entirely of the auricles, and presents the shallow inter-auricular sulcus in which are a branch of the right coronary artery, tributaries of the coronary sinus, and twigs from the right coronary plexus.

The inferior surface, flat and irregularly four-sided, is formed by the right auricle, right ventricle, left auricle, and left ventricle. It is traversed by the auriculo-ventricular sulcus containing the right and left coronary arteries with the coronary sinus, as well as the great, right, and oblique cardiac veins and branches from the right and left coronary plexuses. The posterior inter-ventricular sulcus also intersects this surface, in which runs the descending branch of the right middle coronary artery, with the cardiac vein, and branches from the right coronary plexus.

INTERIOR OF THE HEART.—The four chambers of which the heart is composed vary greatly in form according to the condition of the heart as regards contraction.

The *right auricle* may be regarded as naturally possessing two divisions in free communication with each other by means of an opening admitting the little finger; the one, larger and posterior, termed the sinus venosus or atrium; the other, smaller and projecting forward, known as the auricular appendix. The former portion is irregularly pyriform in shape, with the wider end directed upwards and backwards, and the auricular appendix projects forwards and inwards from the upper part of the anterior aspect. The interior of the atrium is smooth throughout the greater part of its extent, but around the opening into the appendix the surface is marked by the ridges known as the *musculi pectinati*. The interior of the appendix is also furrowed in the same way. The direction of these furrows is obliquely upwards and backwards to their end in the *crista terminalis*.

The posterior wall of the atrium is in great part formed by the inter-auricular septum, and presents, near the entrance of the inferior vena cava, with which indeed it blends, the *fossa ovalis*, partially surrounded above by the *annulus Vieussensii*. This is connected, as will be seen below, with the left end of the Eustachian valve guarding the inferior vena cava. Above the annulus is the tubercle of Lower, varying in prominence in different individuals. Below the *fossa ovalis* is the mouth of the coronary sinus. The *foramina Thebesii*, or openings of the small cardiac veins, are to be seen in different parts of this surface.

The anterior surface presents no feature of interest save the opening into the appendix above, and the pectinated muscles. The superior vena cava enters at the upper and anterior part, and alone among the vessels opening into the auricle is unprovided with a valve. The mouth of the inferior vena cava is at the posterior part of the auricle near the inter-auricular septum, and is guarded by a thin fold of the endocardium, the shrunken remains of the great Eustachian valve of the fœtus. The coronary sinus opens at the lowest part of the posterior wall and is protected by a thin fold of



the lining membrane known as the Thebesian valve. The right auriculo-ventricular orifice is situated at the lowest part



FIG. 1.—Longitudinal section of heart seen from right. The right auricle and ventricle are to the right; the left ventricle and aorta to the left.

of the auricular cavity and is closed by the tricuspid valve, which must be separately described

The interior of the *right ventricle* is of triangular form, when seen in a vertical section whether in an antero-posterior

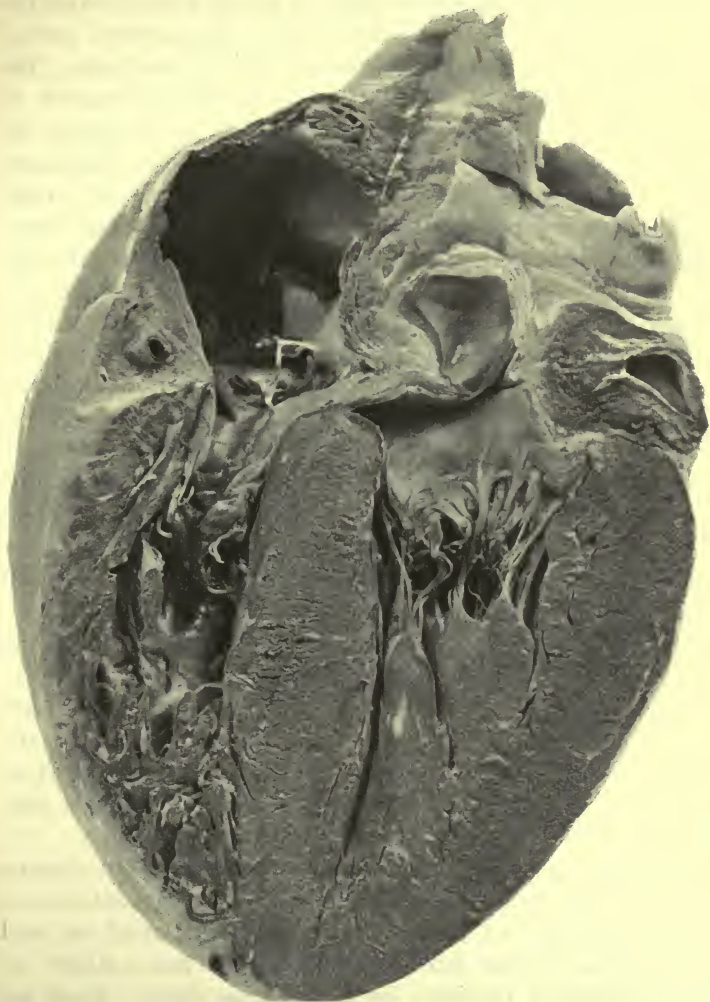


FIG. 2.—Longitudinal section of heart seen from left. The right auricle and ventricle are to the left ; the left ventricle and aorta to the right.

or lateral plane, and has its base towards the auricle. A horizontal section shows the cavity to be crescentic, with the convexity towards the front and right, and the concavity

towards the back and left, on which side it is bounded by the ventricular septum. The left or posterior wall is practically coextensive with the septum, and is much thicker than the right or anterior wall. This latter forms the greater part of the ventricular region of the heart seen from the front. The basal portion of the right ventricle is somewhat irregular, in consequence of being prolonged upwards and to the left as the conus arteriosus. The apex of the right ventricle falls short of the anatomical apex of the heart, and reaches the right border at the junction of the anterior and posterior inter-ventricular sulci. The inner aspect of the wall, excepting the part of the conus arteriosus nearest to the origin of the pulmonary artery, is irregular on account of the columnæ carneæ which project from the interior throughout its whole extent. Some of these are attached to the wall through their entire length, others at both ends, and others only at one end. These latter are aggregated together to form the muscoli papillares. As will be more fully detailed when the construction of the valves is described, these muscoli papillares are usually three in number. A transverse muscular band stretches from the anterior of these papillary muscles across the cavity of the septum, constituting the moderator band.

The *left auricle* is rudely pyriform in shape, with its larger end directed upwards and backwards, and the smaller end downwards and forwards to the left ventricle. At the upper part, towards the anterior and left side, is the auricular appendix, communicating with the atrium by means of an aperture which is smaller than the corresponding one on the right side. The left appendix is less than the right.

The interior of the atrium is smooth except at the opening into the appendix where the muscoli pectinati, which are found in that part of the heart, begin. The part of the wall corresponding to the inter-auricular septum has a slight depression, bounded inferiorly by a crescentic ridge. These are the remains of the foramen ovale and its valve. There are four openings into the left auricle by which the pulmonary veins discharge the blood returning from the lungs. The two bringing the blood from the right lung enter close to the auricular septum; the two from the left lung open near

the appendix. None of these have valves of any kind. The communication between the atrium and appendix is smaller, as has been already mentioned, than the corresponding opening in the right auricle. At the lowest part of the atrium is the left auriculo-ventricular orifice, guarded by the mitral valve.

The *left ventricle* is longer and narrower than the right. Its cavity is triangular when seen in vertical section in any plane, and is circular or oval in a transverse section. The apex of the cavity corresponds to the apex of the heart as a whole, the base is not so oblique as the base of the right ventricle.

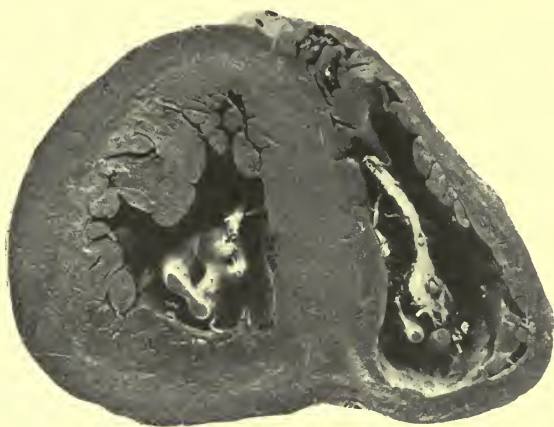


FIG. 3.—Transverse section of heart looking towards apex.

The internal surface is rough and irregular on account of the columnæ carneæ, which are arranged after the same manner as on the right side. The muscoli papillares are thicker than those of the right ventricle; they are usually two in number, but in many hearts there are three.

The general form of the cavities of the heart may be seen in the accompanying illustrations. Figs. 1 and 2 show longitudinal sections through the long axis, about half-way through each ventricle, and give a good idea of the form of each cavity in ventricular systole.

Fig. 3 gives a transverse section through the heart, about half-way between the base and apex of the ventricular portion ;



it brings out the somewhat circular form of the left ventricular cavity, and the crescentic form of the right.

THE ORIFICES AND VALVES OF THE HEART.—The four great cardiac orifices and their valves require separate and special consideration, inasmuch as upon a clear understanding of their mechanism depends a thorough grasp of the central force of the circulation.

The venous orifices, or auriculo-ventricular openings, are placed at the junction of the auricular and ventricular cavities. The right auriculo-ventricular orifice is situated behind and to the right, while the left occupies a position further back and to the left. The aortic orifice occupies the angle in front of these venous openings, and the pulmonary orifice is in the space between the mitral and aortic openings, almost in front of the former.

It must not be forgotten that the venous orifices, although supported as is described elsewhere (p. 43) by cartilaginous rings, are surrounded by muscular fibres, while the arterial openings are simply bounded by the walls of the arteries strengthened by connective tissue.

There is a great difference in the construction of the venous and arterial valves. The former are not only directed downwards, and have their inferior or ventricular surfaces attached to the chordæ tendineæ, but the separate segments are united to each other for a short distance from their connection with the walls of the orifices. The aortic and pulmonary valves are directed upwards, and are quite free at their unattached borders, as well as ununited to each other at the edges of their connection with the vascular walls.

The auriculo-ventricular orifices are both somewhat oval in shape, and in both the longer diameter is approximately transverse. The arterial orifices are almost perfectly circular.

The right auriculo-ventricular, commonly known as the tricuspid, and sometimes as the trochlochine, valve consists almost invariably of three, but occasionally presents four, cusps. The segments are so arranged that one, the largest, is posterior and to the left; it is from its position often termed the septal cusp. Another, in front, is called the infundibular, and the third to the right is known as the external cusp.

These cusps are perfectly smooth on their upper or auricular surface, but the lower or ventricular aspect is broken by the attachments of the chordæ tendineæ, which are not only inserted into the margins, but into different points along the lower surface, and are even, as Noël Paton has recently shown, continued to the ring. The papillary muscles of the right ventricle vary considerably in their origins and attachments. There are three main groups, arising for the most part from the trabecular tissue in the apical part of the ventricle, but they differ somewhat in their connections with the ventricular walls. In some instances they are more closely united to the septum, in others they have more intimate connections with the external wall. When this latter arrangement prevails the moderator band is well developed. The three papillary muscles are: (1) superior, arising below the pulmonary orifice and sending chordæ to the septal and infundibular cusps; (2) anterior, arising from the trabecular tissue, being specially connected with the septum, and giving chordæ to the infundibular and external cusps; (3) posterior, having its origin in the trabecular tissue with especial relation to the outer wall of the ventricle, and sending chordæ to the external and septal cusps. Besides these three great papillary muscles, there are usually some smaller masses projecting from the septum, and sending chordæ to the septal cusp.

The left auriculo-ventricular or mitral valve has its two cusps so placed that one is anterior and to the right, the other posterior and to the left. In general appearance they are similar to the tricuspid segments, but they are much thicker and stronger. The papillary muscles of the left ventricle, two in number, are likewise much larger and stronger than those of the right ventricle. In their arrangement one papillary muscle is placed in front and to the left, the other posteriorly and to the right. Both arise from the external and septal walls of the ventricle, and each has chordæ inserted into both cusps, the anterior papillary muscle being connected with the left edge of the cusps, the posterior with the right.

The circular opening of each arterial orifice is guarded by a valve consisting of three cusps, each of which is in form an equilateral triangle, with the arc of a circle for its base. The

convex base is attached to the walls of the artery; the other borders, which are slightly concave, are free. At the angle formed by the meeting of the free borders is a little nodule named the corpus Arantii. The cusps are strengthened by three bands, one along the attached border, another along the free edge; a third curves from the nodule across the valve, so as to have a crescentic interval, called the lunula, along the edge. Each cusp swells somewhat towards the cavity of the ventricle, and on the wall of the artery opposite each segment is a depression termed the sinus of Valsalva. So far the arrangement is similar in the case of the pulmonary and aortic orifices. The aortic are, however, much stronger than the pulmonary cusps, and the sinuses of Valsalva are in the aorta considerably larger than in the pulmonary artery. The cusps of the pulmonary artery are so arranged that one is posterior and two anterior; those of the aorta are, on the contrary, one anterior and two posterior, of which the one to the left is intimately connected with the anterior mitral cusp.

In relation to two of the aortic cusps are the origins of the coronary arteries. The anterior coronary artery has its origin opposite the anterior cusp, while the posterior coronary artery arises opposite the left posterior segment. It must be observed that the mouths of these vessels are at a higher level than the free edges of the cusps, which seldom cover them.

As was pointed out by Savory, the arterial valves are provided with an important mechanism, by means of which they are supported when bearing the strain of the blood pressure, during the phase of closure. The posterior cusp of the pulmonary valve is placed upon a cushion, formed by the upper part of the ventricular septum, and upon this support the cusp rests when filled with blood. The aortic valve has a similar provision which is much more pronounced. The anterior cusp of this valve is situated above a very prominent cushion, also formed by the ventricular septum, and by its means the valve is aided in withstanding the strain of arterial pressure. As will be shown in another section, the cusps thus supported are those situated at the lowest level and are the first to close during diastole.

THE BLOOD VESSELS OF THE HEART.—The blood supply of the heart is a subject of the greatest importance, for upon its integrity depends the entire energy of the organ; any disturbance of the cardiac circulation inevitably produces more or less interference with its functions.

The two coronary arteries arise from the root of the aorta. The right coronary artery arises from the anterior sinus of Valsalva and passes round in the auriculo-ventricular groove to the posterior aspect of the heart, as far as the inter-ventricular groove, where it meets the left coronary artery. It gives off two main branches, one of which passes down the posterior inter-ventricular sulcus, while the other descends along the right margin of the heart.

The left coronary artery has its origin in the left posterior sinus of Valsalva, and passes behind the pulmonary artery towards the posterior surface of the heart, where it meets the right coronary artery. In its course it gives off a large branch which runs downward in the anterior ventricular groove. The two coronary arteries encircle the base of the heart at the auriculo-ventricular groove, and in their course give off numerous branches to the auricles and the ventricles. The right is the main source of the blood supply to the right auricle and ventricle, while the left may be regarded as almost entirely the source of nutrition for those of the left.

The arrangement of the cardiac veins is by no means similar to that seen in regard to the arteries. It may be said that blood returns from the heart by three channels. The veins which course in the substance of the heart, known as the Thebesian veins, open directly, by several small apertures previously mentioned, into the right auricle. The veins which course upon the surface of the heart terminate in two different ways, the anterior cardiac veins, on the front of the right ventricle, opening directly into the right auricle, while the other superficial veins empty their contents into the coronary sinus. The great cardiac vein arises upon the anterior surface of the heart at the apex, ascends in the anterior inter-ventricular groove to the base, and passes round to the left auriculo-ventricular sulcus to gain the posterior surface of the heart, where it ends in the coronary sinus. In its course it



receives numerous tributaries from the ventricular and auricular portions of the heart, and at its opening into the sinus it is guarded by a bicuspid valve. The right cardiac vein lies in the auriculo-ventricular furrow between the right auricle and right ventricle. It opens into the coronary sinus and is guarded by a valve. There are several posterior cardiac veins, running upon the posterior surface of the heart, ending in the coronary sinus, and guarded in each case by a valve. But one of these which is much larger than the others is termed the middle cardiac vein, and occupies the posterior inter-ventricular groove. One other vein deserves mention on account of two peculiarities. The oblique vein of Marshall, passing along the posterior aspect of the left auricle, opens into the coronary sinus near its termination. Its mouth is not protected by any valve, and it represents the obliterated left superior vena cava of the embryo.

INNERVATION OF THE HEART.—The upper cervical ganglia of the sympathetic, lying between the internal carotid artery and the rectus major muscle, opposite the second and third cervical vertebræ, are connected with the glosso-pharyngeal, pneumogastric, and hypoglossal nerves, as well as with the upper four cervical spinal nerves, and give rise to the superior cardiac nerves. These nerves, which in many instances also receive branches from the sympathetic cord, take the same course on both sides of the neck, behind the carotid sheath, and have connections with the pneumogastric nerves, and its external and recurrent laryngeal branches, but differ in their distribution after entering the thorax. The right superior cardiac nerve passes along the innominate artery, giving small branches to the great vessels in its course, towards the back of the aorta, and ends in the deep cardiac plexus. The left superior cardiac nerve accompanies the left common carotid artery to the arch of the aorta, in front of which it usually passes, to terminate in the superficial cardiac plexus. Occasionally it ends in the deep cardiac plexus instead of taking its usual course. The middle cervical ganglia, lying near the inferior thyroid arteries, have connections with the fifth and sixth cervical spinal nerves, and give rise to the middle cardiac nerves. These nerves pass down behind the carotid sheath

communicating with the recurrent laryngeal and the superior cardiac nerves, and, entering the thorax in close proximity to the subclavian vessel, end in the deep cardiac plexus. The lower cervical ganglia lying between the last cervical vertebrae and the first ribs, receive communicating branches from the two lowest cervical spinal nerves, and give origin to the inferior cardiac nerves. These inferior cardiac nerves pass inwards, communicating with the recurrent laryngeal and middle cardiac nerve, behind the subclavian arteries, to end in the deep cardiac plexus. Sometimes, on the left side, the inferior blends with the middle cardiac nerve.

The vagus nerves also give rise to cardiac branches. The cervical cardiac branches are usually divided into the upper, of which there are several, small in size, ending in the sympathetic cardiac nerves, and the lower, single on each side, of which the right, joining a sympathetic cardiac nerve, ends in the deep, while the left terminates in the superficial cardiac plexus. The thoracic cardiac branches take their origin on the right side from the pneumogastric trunk itself and from the recurrent laryngeal nerve; on the left side they arise from the recurrent laryngeal nerve. These nerves end in the deep cardiac plexus.

The great network known as the cardiac plexus, is, for convenience of description, regarded as consisting of two parts—the superficial and the deep—but these are intimately connected. The superficial cardiac plexus occupies part of the space between the arch of the aorta and the right pulmonary artery. It receives, as we have seen, the left superior cardiac nerve from the sympathetic, and the left lower cervical cardiac branch of the vagus nerve. It furnishes some small branches to the anterior left pulmonary plexus, and terminates in the anterior coronary plexus. The deep cardiac plexus lies between the posterior aspect of the aortic arch and the trachea, and above the bifurcation of the pulmonary artery. With the exception of the two nervous branches ending in the superficial plexus, all the cardiac nerves—sympathetic and pneumogastric—terminate in the deep plexus, which is much larger than the superficial, and is commonly regarded as having a right and left division. It gives off right and left branches. On the right side the plexus

furnishes some filaments to the anterior pulmonary plexus and posterior coronary plexus, together with many branches to the right auricle, but most of the fibres unite with those from the superficial plexus which form the anterior coronary plexus. On the left are some filaments going to the anterior pulmonary plexus, and a few branches to the superficial cardiac plexus, but most of the branches terminate in the posterior coronary plexus.

The anterior coronary plexus, formed by branches from the superficial and both divisions of the deep cardiac plexus, accompanies the left or anterior coronary artery; the posterior coronary plexus, mostly derived from the left, but in part also from the right part of the deep cardiac plexus, follows the right or posterior coronary artery in its course.

These facts may be seen at a glance from the accompanying diagram (Fig. 4), which has been constructed partly from dissections, and partly from the works of Flower, Turner, Cunningham, and Bramwell. The following is the description of the figure.

D, Deep cardiac plexus; S, superficial cardiac plexus; C, coronary plexus; SCG, superior, MCG, middle, and ICG, inferior cervical sympathetic ganglia; SDG, stellate ganglion, and 2-5 DG, other dorsal sympathetic ganglia; AV, annulus of Vieussens; SA, spinal accessory with branches to sterno-mastoid and trapezius muscles, SM and T; V, vagus; GP, glosso-pharyngeal, SL, superior laryngeal, EL, external laryngeal, and RL, recurrent laryngeal nerves; *v, v, v*, branches from vagus to cardiac nerves; *s, s, s*, communicating twigs between sympathetic cardiac nerves; RR, branches to recti; RC, rami communicantes; SO, small occipital; GA, great auricular; CL, clavicular; ST, sternal; AC, acromial; P, phrenic; SS, suprascapular; SCL, subclavian; ER, nerve of Bell; EAT, external anterior thoracic; SSS, subscapular; MC, musculo-cutaneous; M, median; MS, musculo-spiral; U, ulnar; IC, internal cutaneous; LIC, lesser internal cutaneous; ICH, intercosto-humeral. The spinal cord is represented as divided into two lateral halves, right and left; the Roman numerals denote the segments corresponding to the eight cervical and upper dorsal nerves, whose anterior and posterior roots are shown, the latter characterised by their ganglia; the connections between the spinal segments and sympathetic ganglia are marked  $c^1$ - $c^8$  and  $d^1$ - $d^5$ .

**DIMENSIONS OF THE HEART.**—Many discrepancies will be found in the statements made by different authors with reference to the weights and measurements of the healthy heart. The figures given by Peacock, Reid, Clendinning, Rankine, and

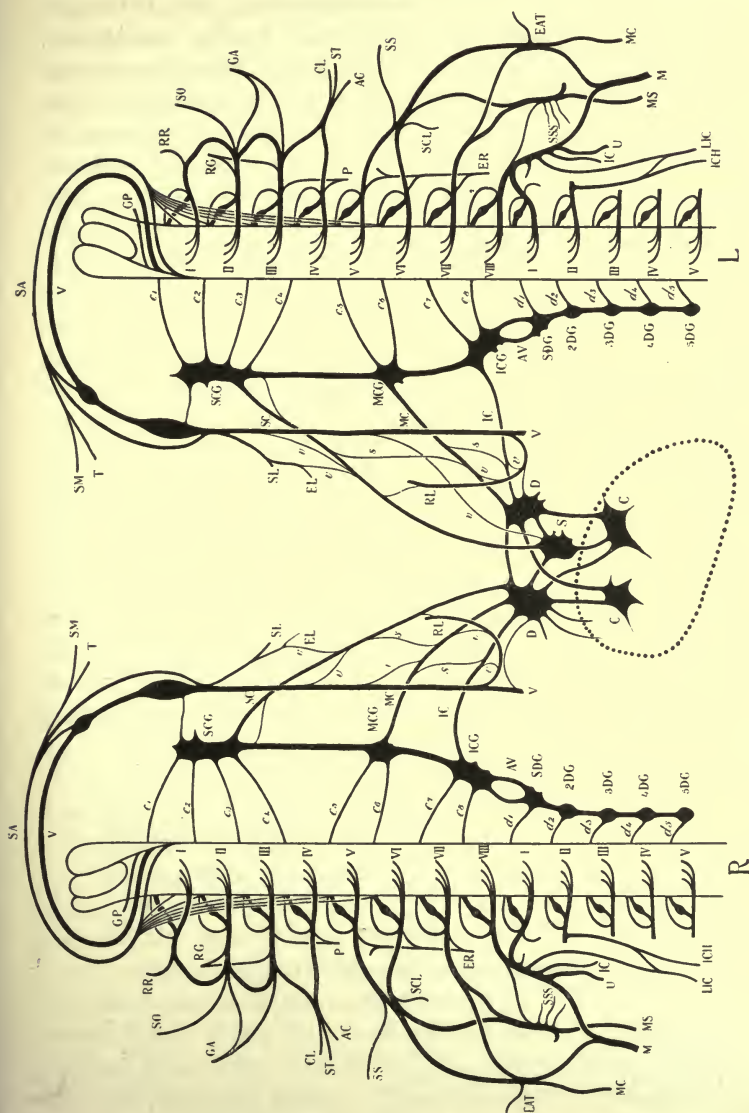


FIG. 4.—Diagram of the connections of the cardiac nerves. For description see text, p. 24.



Bizot differ widely, and the explanation of the want of correspondence between their statements can only be sought in a possible want of care in selecting healthy individuals. The most recent facts are those of Hamilton, from observations upon healthy individuals who met their death suddenly from traumatic causes. He finds that the weight of the heart in men varied between 10 and 16 oz., with an average of 10 to 13 oz. In women the weight ranged from 7 to  $15\frac{1}{2}$  oz., and its commonest weight was from 10 to 11 oz. His results show, as was previously suspected, that there is no real ratio between the size of the body and that of the heart; yet they indicate that, as a rule, the heaviest hearts are to be found in the tallest persons. Observations on the length of the ventricular cavities, estimated by measuring the distance from the apex of the cavity to the base of the nearest valvular cusp, show that the left ventricle varies in men from  $2\frac{1}{2}$  to  $3\frac{3}{4}$  in., with an average of  $3\frac{1}{4}$  in., and in women from  $2\frac{1}{4}$  to  $3\frac{1}{2}$  in., with an average of 3 in. The right ventricle in men was from 3 to 4 in. in length, giving a mean of  $3\frac{3}{8}$  in.; and in women from 3 to  $3\frac{1}{4}$  in., with an average of  $3\frac{1}{16}$  in.

The thickness of the wall of the left ventricle is about  $\frac{1}{4}$  in. at the apex and  $\frac{1}{2}$  in. at the base in both sexes, while that of the right ventricle is, in every case on an average,  $\frac{1}{8}$  in. all over in both sexes. The diameters of the various orifices are given by Hamilton in the following table:—

## DIAMETERS OF ORIFICE—MALE.

	Greatest.	Least.	Average.
Aortic . . . .	1·3 in.	·9 in.	1 in.
Mitral . . . .	1·8 „	1·1 „	1·4 „
Pulmonary . . .	1·5 „	1 „	1·2 „
Tricuspid . . .	2·2 „	1·3 „	1·8 „

## DIAMETERS OF ORIFICE—FEMALE.

	Greatest.	Least.	Average.
Aortic . . . .	1 in.	·8 in.	·9 in.
Mitral . . . .	1·5 „	1 „	1·2 „
Pulmonary . . .	1·3 „	1 „	1·1 „
Tricuspid . . .	1·7 „	1·4 „	1·5 „

POSITION AND RELATIONS OF THE HEART AND GREAT VESSELS.—The position of the heart and great vessels in the

thorax, and their relations to the other viscera of the chest, are subject to great variations even in conditions of health; from the general average of observations, nevertheless, a certain mean is obtained, which may be considered as the standard. In the present section of the work this standard will be described, and changes in the position and relations of the central organs of the circulation will be discussed elsewhere.

The heart occupies the space in the thoracic cavity between the anterior and the posterior mediastinum, but, according to the age of the individual and the form of the thorax, its position may be very different. The anterior mediastinum, or the space between the sternum in front, the pericardium behind, and the two pleuræ on either side, contains the triangularis sterni muscle, and the origin of the sterno-hyoid and sterno-thyroid muscles, along with the remains of the thymus gland and some areolar tissue. The posterior mediastinum, bounded in front by the pericardium and the roots of the lungs, behind by the vertebral column, and laterally by the two pleuræ, has for contents the thoracic duct, the vena azygos, the œsophagus, the trachea, and the aorta, as well as the sympathetic and splanchnic nerves. The intervening space between these spaces, commonly known as the middle mediastinum, is occupied by the heart and its great vessels. The heart with the origins of the great vessels may, therefore, be said to be embraced by the lungs, the sternum, and the spine. It is necessary, however, to look more closely into its relations, and the best method of doing so is to consider those relations from above downwards.

Starting with the highest point of the arch of the aorta, there are in front the sterno-thyroid and triangularis sterni muscles, the two pleuræ, a small portion of each lung, and some areolar tissue. The internal mammary vessels, as a rule, lie external to the aorta and superior vena cava. Somewhat lower down the internal mammary vessels become approximated more closely to the mesial plane, and lie in front of the great vessels. The two pleuræ with their lungs meet near the left edge of the sternum, so as almost entirely to cover the great vessels in front, and, about the level of the pulmonary orifice, the right auricular appendix curves

round so as to be almost in front of the pulmonary artery. The same relationship persists until reaching the level at which the left pleural sac with its contained lung turns out; in front of the heart below this level there is a considerable space altogether uncovered by lung.

Posteriorly, the great vessels have behind them some areolar tissue, the trachea, the œsophagus, the azygos vein, and the sympathetic nerves. Just behind the trachea lies the trunk of the right vagus nerve, and at the left edge of the trachea, between it and the aorta, is the recurrent laryngeal branch of the left vagus nerve. At a lower level the heart is in front of the œsophagus, the two bronchi, the azygos vein, and the sympathetic nerves, while the aorta lies behind the left bronchus. Somewhat further down, below the level of the bronchi, there are, posteriorly, from right to left, the œsophagus, the vena azygos, and the aorta. The left vagus lies between the heart and the vena azygos, and the right vagus lies in the angle formed by the heart and the œsophagus. At a still lower level the heart has behind it the liver, and behind that viscus are the œsophagus, the aorta, and the vena azygos; here the left vagus lies between the œsophagus and the liver, the aorta between the œsophagus and the vena azygos. The left phrenic nerve lies in the angle formed by the liver and the œsophagus, while the right phrenic nerve lies between the liver and the posterior edge of the right lung.

On the right side of the great vessels above is the right lung with its pleura. Between the superior vena cava and the lung lies the right phrenic nerve. The same relationship is preserved on descending to a lower level, but the phrenic nerve inclines somewhat further towards the back.

On the left side, from above downwards, are the left lung with its pleura, and between the lung and the ascending aorta is placed the phrenic nerve, while between the lung and the descending aorta is placed the left vagus nerve. At a little lower level the left auricular appendix comes round to the left side of the pulmonary artery. The phrenic nerve lies in the pleuro-pericardium between the lung and the auricular appendix. Still lower the pleuro-pericardium and the lung

form the entire boundary of the heart, with the left phrenic nerve lying between the serous layers, and, at the lowest point, there is nothing to the left of the heart excepting the pleuro-pericardium and the left lung.

The consideration of longitudinal sections through the thorax leads to most useful information as regards the relations of the heart and neighbouring structures within the thorax. The three following illustrations are reproductions of photographs obtained by the kindness of Professor Cunningham



FIG. 5.—Sagittal section through the thorax of a male adult at a point half an inch inside the mid-Poupart plane. *n*, Left lung; *o*, left ventricle of the heart; *p*, liver; *q*, spleen.

from sections of a male adult in his possession, and show some of the most important relations of the heart and other thoracic viscera. Fig. 5, obtained at a point half an inch to the inside of the mid-Poupart plane, shows the heart lying within the pericardial sac and surrounded for about three-quarters of its circumference by the lung tissue. In front, a part of the heart corresponding to the superficial cardiac dulness is seen to be uncovered by the lung, and below, half of the inferior surface of the heart at this level has also no



relations to the lung, being only separated from the liver by the pericardium, the diaphragm, and the peritoneum.

Fig. 6, obtained at a point one quarter of an inch to the left of the left edge of the sternum, shows that in a plane close to the vertebral column and the edge of the sternum the heart and great vessels occupy the entire antero-posterior diameter of the chest. The section shows part of the right ventricular cavity, terminating in the conus arteriosus and the pulmonary artery, the right branch of which is seen passing

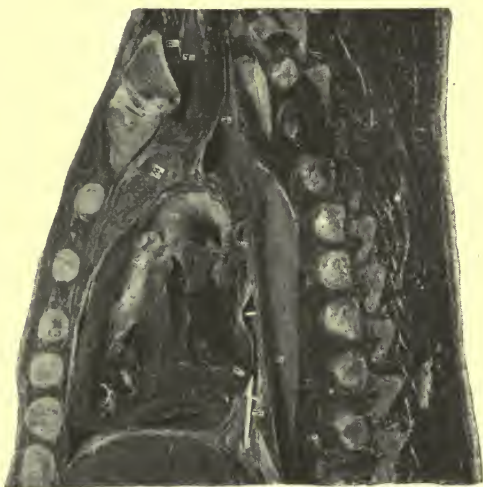


FIG. 6.—Sagittal section through the thorax of a male adult at a point one-quarter of an inch to the left of the left margin of the sternum. *g*, Vena azygos major; *h*, aorta; *j*, left bronchus; *k*, coronary sinus; *l*, left auricle; *m*, left ventricle; *n*, right ventricle; *o*, pulmonary artery; *p*, left subclavian artery; *q*, left common carotid artery; *r*, left internal jugular vein; *s*, sterno-hyoid and sterno-thyroid muscles; *w*, remains of thymus gland.

over to its proper side. The inferior extremity of the infundibular cusp of the tricuspid valve and its chordæ may be observed towards the lower portion of the ventricular cavity, and the pulmonary cusps mark the termination of the conus and the commencement of the artery. Behind the right ventricle is a portion of the left ventricular cavity running upwards in the direction of the origin of the aorta. At a level above the pulmonary artery is seen the arch of the aorta

passing backwards and downwards as it comes over from the right side to become the descending aorta. It is closely applied to the heads of the ribs. Between the aorta and the posterior wall of the heart is the œsophagus, and between the aorta and the pulmonary artery is the left bronchus. In front of the heart in its upper part is a small portion of the

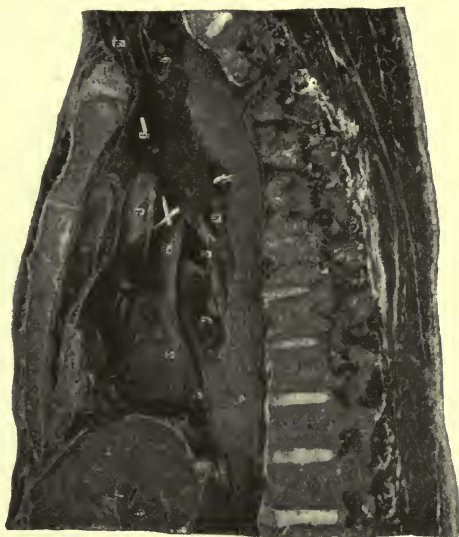


FIG. 7.—Sagittal section through the thorax of a male adult immediately to the right of the mesial plane. *b*, Liver; *o*, right auricle (the letter is at the edge of the crista terminalis); *d*, inferior vena cava; *e*, right lung behind the auricle; *f*, right pulmonary veins; *g*, right pulmonary artery; *h*, right bronchus; *i*, greater azygos vein; *j*, left innominate vein; *l*, innominate artery; *m*, superior vena cava (the directors are pushed upwards into the innominate and azygos veins); *r*, pericardium in front of superior vena cava; *s*, lung in front of great vessels.

left lung, while below it is the liver. Fig. 7 is taken slightly to the right of the mesial plane. It shows the atrium of the right auricle with which the superior and inferior venæ cavæ communicate, and upon the walls of which are the pectinated muscles ending in the crista terminalis. A portion of the auricular appendix is seen projecting upwards into the upper part of the pericardial sac. In front of the upper portion of the auricle there is a small part of the right lung, while a

considerable thickness of the right lung is seen between the heart and its vessels and the vertebral column. Between the superior vena cava and the lung are the vena azygos major, the right bronchus, the right pulmonary artery, and the right pulmonary veins. Below the heart is the liver.

The study of transverse sections of the thorax affords equally important information as to the relations of the heart. These may be seen in the accompanying illustrations obtained by the kindness of Professor Symington from sections of a

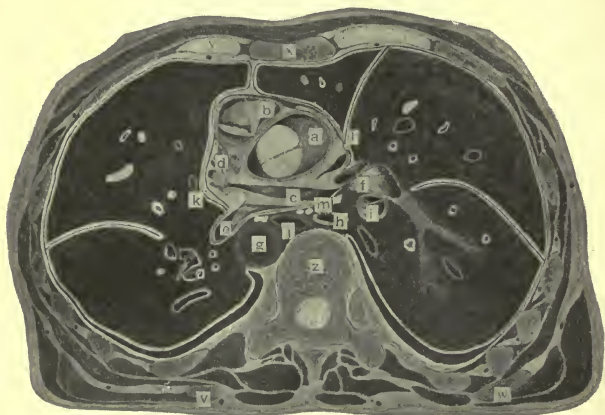


FIG. 8.—Section through eighth dorsal vertebra. *a*, Ascending aorta; *b*, pulmonary artery; *c*, right auricle; *d*, auricular appendix; *e*, pulmonary vein; *f*, branch of pulmonary artery; *g*, descending aorta; *h*, vena azygos; *i*, bronchial tube; *j*, cesophagus; *k*, left phrenic nerve; *l*, right phrenic nerve; *m*, vagus and sympathetic nerves; *x*, sternum; *y*, third costal cartilage; *z*, eighth dorsal vertebra.

male adult in his possession. As the subject was 57 years old, the position of the viscera is necessarily somewhat lower than in a younger man. Fig. 8 is at the level of the eighth dorsal vertebra, and the third costal cartilage. It shows the pulmonary artery so close to the valve that the right cusp has been cut across, and the ascending aorta somewhat further from its valve. Behind these great vessels is the left auricle, into which two of the pulmonary veins may be seen entering, while the superior vena cava lies in the angle between the aorta and the auricle. The descending aorta, azygos vein, and cesophagus are seen behind the auricle, with the vagi and



sympathetic nerves in the intervening space. The phrenic nerves lie between the surrounding lungs and the roots of the

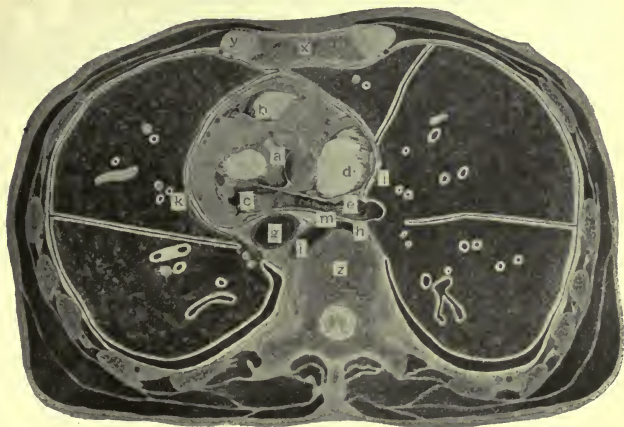


FIG. 9.—Section through ninth dorsal vertebra. *a*, Left ventricle with aortic cusps; *b*, conus arteriosus; *c*, left auricle; *d*, right auricle; *e*, pulmonary vein (right); *g*, descending aorta; *h*, vena azygos; *j*, œsophagus; *k*, left phrenic nerve; *l*, right phrenic nerve; *m*, sympathetic and vagus nerves; *x*, sternum; *y*, fourth costal cartilage; *z*, ninth dorsal vertebra.

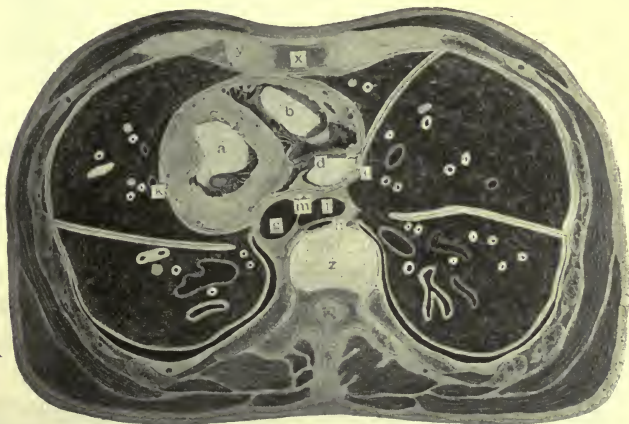


FIG. 10.—Section through disc between ninth and tenth dorsal vertebra. *a*, Left ventricle; *b*, right ventricle; *d*, right auricle; *g*, descending aorta; *h*, vena azygos; *j*, œsophagus; *k*, left phrenic nerve; *l*, right phrenic nerve; *m*, vagus and sympathetic nerves; *x*, sternum; *y*, fifth costal cartilage; *z*, disc between ninth and tenth dorsal vertebra.

aorta and pulmonary artery in the pleuro-pericardium. Fig. 9 is taken at the level of the ninth dorsal vertebra and fourth

costal cartilage. The section shows the conus arteriosus in front, while the root of the aorta is cut through so near the level of the arterial valve that the left posterior cusp has disappeared, while the right and anterior cusps remain. The right auricle is shown to the right of the aortic cusps, and the left lies behind. Fig. 10 gives a representation of the relations at the level of the disc between the ninth and tenth dorsal vertebræ and of the fifth costal cartilage. At this point the right auricle is seen opening into the ventricle, and septal and external cusps of the tricuspid valve lie between the two cavities; the left ventricle is cut across immediately above the papillary muscles, the right of which may be seen with the cut ends of the chordæ tendineæ.

**SURFACE RELATIONS.**—The heart is so placed that its long axis is at an angle of about  $60^\circ$  to the long axis of the body. The base of the heart is directed upwards and to the right; the apex downwards and to the left. When the limits of the heart are projected upon the surface of the body, they are found to exhibit many individual variations. But amongst these a certain average is found to obtain, which may be taken as the healthy standard.

*Position and limits of the Heart.*—The highest point which the heart reaches is at the upper edge of the third left costal cartilage, close to the sternal margin, and from this point the cardiac boundaries run outwards and downwards on both sides. The base, formed by the termination of the conus arteriosus of the right ventricle, and by the right auricle with its appendix, starting from the point just mentioned, crosses the sternum to the right articulation between it and the third rib. The right edge, formed by the right auricle, runs downwards and slightly outwards until it reaches the fifth costal cartilage, about an inch and a half from mid-sternum. The left edge, formed by the left ventricle, extends from the upper level of the third left costal cartilage, running considerably outwards as well as downwards. It crosses the third costal cartilage, the third interspace, the fourth rib, the fourth interspace, the fifth rib, and terminates in the fifth intercostal space about  $3\frac{1}{2}$  inches from the mid-sternal plane. The inferior edge, which is formed by the right ventricle and the apical portion

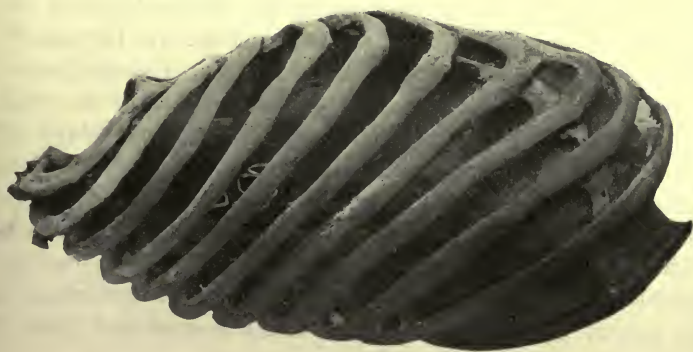


FIG. 11.—Heart in thorax seen from the right side.

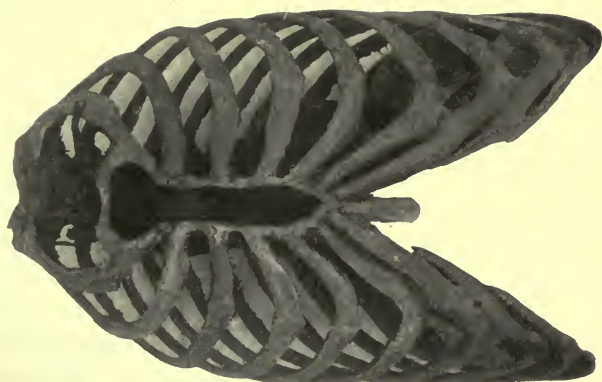


FIG. 12.—Heart in thorax seen from the front.

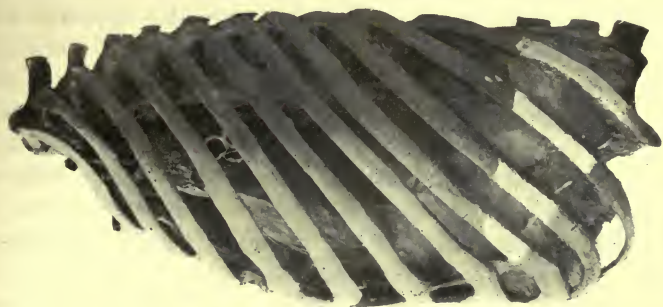


FIG. 13.—Heart in thorax seen from the left side.



of the left, extends from the fifth right costal cartilage to the fifth left intercostal space. The right auricular appendix lies behind the right half of the sternum at the level of the third costal cartilage. The left auricular appendix occupies the second left intercostal space on the outer aspect of the pulmonary artery. The four great cardiac orifices, although varying considerably in position, present an average relation to the surface in the healthy adult. The pulmonary orifice is found at the lowest level of the second left intercostal space, and the adjoining portion of the sternum. It is so situated that its axis is directed upwards, backwards, and to the left. The aortic orifice is underneath the sternum, its junction with the third left costal cartilage, and a small part of the second left intercostal space. Its axis is directed upwards, backwards, and to the right, and about one-quarter of the orifice is covered by the pulmonary orifice. The mitral orifice lies below the left half of the sternum, at the level of the third intercostal space and fourth costal cartilage. Its axis is directed upwards, backwards, and to the right. The tricuspid orifice is underneath the left half of the sternum, from the fourth to the fifth intercostal space. Its axis is also directed upwards, backwards, and to the right. It is to be remarked that the mitral and tricuspid orifices have but slight direction upwards; their main axis is to the right and slightly to the back. The tricuspid orifice, in some instances, crosses the mesial plane, so that it may in part lie underneath the right half of the sternum. The pulmonary and tricuspid orifices are superficial, and lie comparatively near the chest wall, but the aortic and mitral orifices are much more deeply placed. The relation of the heart to the thoracic parietes is shown in Figs. 11, 12, and 13, from a specimen in the possession of Professor Cunningham.

*Position of the Large Vessels.*—The aorta takes its origin behind the left half of the sternum, opposite the third left costal cartilage, and it runs upwards and to the left, with a slight inclination forwards, until it reaches the second right costal cartilage, from which point it runs inwards and backwards behind the sternum, which it crosses at the level of the first intercostal space. The pulmonary artery has its origin in the second left intercostal space at its sternal end, and in



this interspace it runs outwards and backwards for about one inch.

The right innominate vein has its origin behind the sternal end of the right clavicle, and runs almost vertically downwards to meet the corresponding vein from the left side.

The left innominate vein, lying immediately above and in contact with the arch of the aorta, begins behind the sternal end of the left clavicle and lies behind the upper part of the manubrium sterni; it joins the right innominate vein behind the first right chondro-sternal articulation.

The superior vena cava lies to the right side of the arch of the aorta behind the first and second right cartilages and the intervening intercostal space, and terminates behind the second intercostal space.

In relation to the back of the chest, the heart occupies a position in front of the 5th, 6th, 7th, and 8th dorsal vertebræ. The highest part of the arch of the aorta is at the level of the 3rd dorsal vertebra. It reaches the spine at the 4th, and becomes the descending aorta at the 5th, dorsal vertebra. The bifurcation of the pulmonary artery takes place at the level of the 4th dorsal vertebra, at which point also is the bifurcation of the trachea. The great thoracic landmarks may be best summarised in such a table as that which follows on next page.

The heart is enveloped by the lungs throughout almost its entire extent, only a small portion of its anterior surface being uncovered by them. The anterior margins of the lungs, approaching each other as they descend, almost meet behind the left half of the sternum opposite the 1st intercostal space. From this point the two margins descend side by side, and the right lung continues its course downwards until it reaches the 6th costal cartilage, at which point it turns outwards while still continuing its downward course, and obliquely crosses the 6th intercostal space, the 7th rib, the 7th intercostal space, and in the mid-axillary line is at the level of the 8th rib. Proceeding backwards this margin crosses the 8th intercostal space, the 9th rib and interspace, the 10th rib and interspace, and reaches the spine posteriorly at the level of the 10th dorsal vertebra about its upper end.

## LUNGS.

## ANTERIORLY.

Apices of Lungs 1-1½ inches above clavicles ; Right apex usually a little higher than Left.

Lungs meet opposite manubrio-gladiolar articulation, and diverge at 4th costal cartilage ;  
 Right Lung turns *out* at 6th chondro-sternal joint ;  
 crosses 6th and 7th intercostal spaces ;  
 reaches 8th *rib* in axillary line.

Left Lung turns *out* at 4th costal cartilage ;  
 crosses 4th intercostal space ;  
 turns *in* again at 5th costal cartilage ;  
 crosses 5th intercostal space ;  
 turns *out* finally at 6th costal cartilage ;  
 crosses 6th and 7th intercostal spaces ;  
 reaches 8th *intercostal space* in axillary line.

Upper and lower Lobes separated by line from 2nd dorsal spine to 6th rib.  
 Middle and lower Lobes separated by line from middle of above line to 4th chondro-sternal joint.

Upper and lower Lobes separated by line from 2nd dorsal spine to 6th rib.

## POSTERIORLY.

Right Lung reaches *upper* border of 10th dorsal vertebra.

Left Lung reaches *lower* border of 10th dorsal vertebra.  
 Bifurcation of Trachea at level of 4th dorsal vertebra.

## HEART.

## ANTERIORLY.

Base of Heart extends from lower border of 2nd costal cartilage of left side to upper border of 3rd costal cartilage of right side.  
 Apex reaches 5th left intercostal space.

Aorta arises at level of 3rd costal cartilages behind sternum ;  
 reaches half-way up manubrium sterni ;  
 is nearest surface at 2nd right costal cartilage.

Pulmonary Artery arises in lower part of 2nd left intercostal space close to sternum ;  
 ends behind the 2nd left costal cartilage ;  
 is nearest surface at 2nd left intercostal space.

Valves. Aortic behind junction of 3rd *left* costal cartilage with sternum (one-fourth is covered by pulmonary valves).  
 Pulmonary behind upper border of 3rd *left* costal cartilage, and adjacent part of 2nd left intercostal space.  
 Mitral behind *left* half of sternum at 3rd intercostal space and 4th costal cartilage.  
 Tricuspid behind *left* half of sternum at 5th costal cartilage and 4th intercostal space.

## POSTERIORLY.

Base of Heart at level of 5th dorsal vertebra.

Apex at level of 8th dorsal vertebra.

Aorta reaches spine at level of 4th dorsal vertebra.

Pulmonary Artery bifurcates at level of 4th dorsal vertebra.

## CLINICAL REGIONS AND THEIR CONTENTS.

## ANTERIORLY.

RIGHT SIDE—		LEFT SIDE—	
Supra-clavicular. <i>Lung, Subclavian Vessels, Carotid Vessels.</i>	Supra-sternal <i>Trachea.</i>	Supra-clavicular. <i>Lung, Subclavian Vessels, Carotid Vessels.</i>	Supra-clavicular. <i>Lung, both Ventricles, Part of Left Auricle, part of Pulmonary and Aortic Valves.</i>
Infra-clavicular. <i>Lung, Vena Cava Superior, Right Auricle.</i>	Superior Sternal. <i>Lungs, Arteria Innominate, Vena Innominate, Vena Cava Superior, Aorta, Arteria Pulmonalis, Right Auricle and Ventricle, Part of Every Valve.</i>	Infra-clavicular. <i>Lung, both Ventricles, Part of Left Auricle, part of Pulmonary and Aortic Valves.</i>	Infra-clavicular. <i>Lung, both Ventricles, Part of Left Auricle, part of Pulmonary and Aortic Valves.</i>
Mammary. <i>Lung, Liver, Right Auricle.</i>	Inferior Sternal. <i>Part of Right Auricle and Ventricle, Part of Tricuspid Valve. Lungs, Liver.</i>	Mammary. <i>Part of Lung, both Ventricles, Stomach.</i>	Mammary. <i>Part of Lung, both Ventricles, Stomach.</i>
Infra-mammary. <i>Liver, Lung.</i>	Infra-mammary. <i>Lung, Stomach, Spleen.</i>	Infra-mammary. <i>Lung, Stomach, Spleen.</i>	Infra-mammary. <i>Lung, Stomach, Spleen.</i>
Axillary. <i>Lung, Bronchus.</i>	Axillary. <i>Lung, Bronchus.</i>	Axillary. <i>Lung, Bronchus.</i>	Axillary. <i>Lung, Bronchus.</i>
Infra-axillary. <i>Lung, Liver.</i>	Infra-axillary. <i>Lung, Liver.</i>	Infra-axillary. <i>Lung, Liver.</i>	Infra-axillary. <i>Lung, Liver.</i>

## POSTERIORLY.

Supra-spinous. <i>Lung.</i>	Supra-spinous. <i>Lung.</i>	Supra-spinous. <i>Lung.</i>	Supra-spinous. <i>Lung.</i>
Infra-spinous. <i>Lung.</i>	Infra-spinous. <i>Lung.</i>	Infra-spinous. <i>Lung.</i>	Infra-spinous. <i>Lung.</i>
Infra-scapular. <i>Lung, Liver, Kidney, Intestines.</i>	Infra-scapular. <i>Lung, Liver, Kidney, Intestines.</i>	Infra-scapular. <i>Lung, Liver, Kidney, Intestines.</i>	Infra-scapular. <i>Lung, Liver, Kidney, Intestines.</i>

The anterior margin of the left lung turns out at the level of the 4th costal cartilage, and obliquely crosses the 4th intercostal space and the 5th rib. But at this point it usually turns in again until it reaches the 6th rib, when it turns outwards once more and crosses the 6th intercostal space, the 7th and 8th ribs and interspaces, so as to reach the 8th intercostal space in the mid-axillary line. It then crosses the 9th and 10th ribs and interspaces, and reaches the spine at the lower level of the 10th dorsal vertebra. It thus leaves a somewhat rudely triangular part of the anterior aspect of the heart uncovered.

It is usual to divide the thorax into certain conventional areas, by means of arbitrary vertical and horizontal lines. Of the vertical lines one may be drawn corresponding to the mesial plane, but it is more general to draw two parallel lines, one along each edge of the sternum as far as the costal attachments. Two vertical lines are also drawn through the middle point in Poupart's ligament, which is termed the mid-Poupart plane. Two other vertical lines are drawn at the level of the anterior fold of the axilla, two others at the posterior fold of the axilla, and two more vertically drawn through the innermost portions of the posterior edge of the scapulæ. The horizontal lines are : one drawn at the level of the clavicle at its inner end, a second at the level of the lowest part of the third rib, another at the tip of the xiphoid process of the sternum, and another at the lowest point of the costal margin. These arbitrary lines give respectively the clavicular, the mammary, the xiphoid, and the sub-costal planes, and subdivide the thorax into a number of conventional areas. The anterior areas are : in the middle line, the superior and inferior sternal ; laterally, above the clavicles, the supra-clavicular areas ; below the clavicles, the infra-clavicular and the mammary ; the axillary, and the infra-axillary. Posteriorly, the areas are : the supra-spinous, the infra-spinous, the inter-scapular, and the infra-scapular.

The contents of these different areas, that is to say, the viscera which lie underneath them, can be best expressed in such a table as that which is subjoined.

The modern development of photography by means of the x-rays has permitted the relations of the heart to the skeleton



to be clearly made out. This subject will be taken up under Symptomatology.

STRUCTURE OF THE HEART.—A consideration of the structure of the heart naturally falls into divisions corresponding to the layers of which it is composed. It is also necessary to make reference to its more special adaptations to particular uses, and to give some consideration to the special facts of vascular and nervous supply.

*The Pericardium.*—The parietal layer of the pericardium is, in its structure, a strong tough membrane composed of interlacing fibres, containing much yellow elastic tissue, lined on its inner aspect by a layer of flattened polygonal cells, which are nucleated. The lining, or serous layer, is reflected over the heart. The only point of distinction between this serous membrane and similar structures elsewhere is that no lymphatic apertures have yet been found.

*The Endocardium.*—This consists of a single layer of flat polygonal endothelial cells lying upon a thin elastic layer. Underneath the elastic layer is a stratum of nucleated white fibrous tissue, connected with the myocardium by loose areolar bundles and yellow elastic fibres. There are also some muscular fibres, which resemble the general muscular tissue of the myocardium, and some fat. The relative proportion of these different elements varies in the different regions of the heart, the elastic fibres, for instance, being more fully developed in the auricles than in any other part.

*The Myocardium.*—The myocardium, or muscular layer, has for its structural basis a nucleated columnar cell, with processes, and without sarcolemma. The cell is united by cement at its ends to similar cells in such a way as to form a striated fibre, while its processes join others and give rise to a network. Amongst these muscular elements there is a ramification of connective tissue, bearing blood and lymph vessels, and the myocardium is so liberally supplied with both, but particularly with the latter, that it has been aptly compared to a sponge. The muscular tissue of the heart is not continued along the great arterial trunks, but spreads along the great veins, *venæ pulmonales* as well as *venæ cavæ*, which open into the auricles.

*Arrangement of Muscular Fibres.*—The method of arrangement of the muscular fibres of the heart was carefully studied by Lower, who clearly made out their somewhat spiral course. His work is illustrated by figures which must infallibly arouse the admiration of any one who takes the trouble of studying them. Senac devoted a considerable amount of attention to this subject, and the portion of his work dealing with the structural arrangements of the muscular fibres is also well worthy of perusal. A great advance was made by Pettigrew, who, with painstaking diligence, investigated the different layers into which the muscular fibres resolve themselves, and who was able to make out that these may be separated into seven distinct layers. More recently the subject has been again studied by Hesse, Krehl, and Romberg. Their observations have been of service in leading to a clearer understanding of the structure of the heart.

The structural basis of the left ventricle is a circular arrangement of muscle fibres, consisting of several layers which intermingle to a considerable extent, but form a closed circuit. In this way is produced the greatest part, not only of the outer wall of the left ventricle, but of the inter-ventricular septum. This circular muscle, as it may be called, has an opening at the base and another at the apex, and it is covered externally by muscular fibres, which have their origin in the region of the auriculo-ventricular sulcus. The latter pursue a course downwards to the apex, and pass through the opening in the circular muscle at that part in order to gain its inner surface, up which they proceed, giving origin to the papillary muscles and columnæ carneæ in their course towards the auriculo-ventricular region. The circular muscle is therefore embraced, as it were, by these longitudinal fibres, and for practical purposes the left ventricle may therefore be regarded as composed of three layers, separated, but yet closely united by fibres passing from one to the other, more particularly about the region of the papillary muscles. The structure of the right ventricle is quite different, and it may be regarded as only possessing two distinct layers. The outer of these is formed by fibres, which, arising from the posterior part of the left ventricle and from the base of the heart, run downwards



towards the apical region of the heart. Within this layer is another running, for the most part, from the base to the apex of the heart, parallel, for the most part, to the long axis of the ventricle. This inner layer forms the largest portion of the right ventricle. The papillary muscles of the right ventricle have their origin in fibres connected with both layers coming from all points.

*The Orifices and Valves.*—The orifices of the heart are supported by processes extending from the central mass of fibro-cartilage placed in the space between the two auriculo-ventricular and the aortic orifices, which are continued into the fibrous rings surrounding the auriculo-ventricular and arterial orifices. The fibrous rings are distinct from each other, except in front of the mitral orifice, where they are connected. Both the auriculo-ventricular and semilunar cusps are duplications of the endocardium, containing a large amount of elastic tissue, but there are differences in structure between the venous and arterial cusps. In the former the fibrous tissue ramifies in every direction; in the latter it is collected in the bands already described. The former also contain a considerable amount of striated muscular tissue arising from the auricular myocardium, and extending about a third of the distance from the attachment of the cusp to its free margin. During foetal life, according to Langer, there is much more muscle in these valves. Muscular tissue is entirely absent from the semilunar cusps. Luschka described both auriculo-ventricular and semilunar valves as being abundantly supplied with blood vessels; but, according to the researches of Langer and Coen, there is a great difference between the two series of valves in regard to vascular supply. The auriculo-ventricular cusps are supplied, at any rate in the upper part, by blood vessels, which seem to accompany the muscular fibres. The semilunar cusps, on the other hand, are destitute of blood vessels, so that, like the greater part of the endocardium, they must be nourished simply by lymph vessels, with which they are well supplied. According to Luschka and Darier fine prolongations of the arterial system of the papillary muscles are continued along the chordæ tendineæ.

THE BLOOD AND LYMPH VESSELS. — The blood vessels,

which supply the heart, are abundantly distributed throughout the interstices of the myocardium in every part. Lymph vessels are also widely spread throughout the tissues of the heart, especially in the sub-endocardial and sub-pericardial substance, and appear to arise in the form of spaces lined by epithelioid cells.

*The Vessels.*—In order to complete this brief sketch of the leading anatomical facts of the circulation, a rapid glance may be given to the structure of the vessels engaged in the conveyance of the blood and lymph.

The structure of the arteries is much more complicated than that of the veins or capillaries. The arterial wall consists of three more or less distinct layers. The inner coat, or tunica intima, is composed internally of a layer of flattened fusiform cells longitudinally arranged, on the outside of which there is a fenestrated basement membrane, varying in thickness with the size of the vessel, and commonly known as the internal elastic lamina. The middle coat, or tunica media, consists of one or more layers of spirally arranged muscular fibres. In the larger vessels, where there are several strata of muscular fibres, these are separated by layers of connective tissue. The outer coat, or tunica adventitia, often also known as the tunica extima, is composed of bundles of nucleated connective tissue fibres longitudinally arranged, and of yellow elastic fibres. This outer coat blends with the surrounding connective tissue in the case of the smaller vessels, and in the case of the larger arteries it is protected by a distinct sheath. The arteries are provided with their own nutritive arteries, the vasa vasorum, which proceed inwards from the outer layers as far as the middle coat.

The walls of the capillaries are composed of fusiform flattened cells, provided with nuclei, longitudinally arranged and cemented together. They lie in the connective tissue, and are surrounded by lymph spaces.

The structure of the veins is somewhat similar in the main to that of the arteries, and the venous wall also shows three layers, which are not, however, so distinct as those seen in the arteries. The inner coat has a layer of flat polygonal cells, and another of fine fibres which are felted together. The

middle coat consists of white connective tissue, with a few elastic fibres, and an extremely variable amount of muscular tissue. In some veins, as those of the brain, there are few, if any, muscular fibres; in others, such as the venæ cavæ and portal vein, muscular tissue is abundant. The outer coat is composed of white and yellow fibres, with scattered muscular tissue in some cases. The veins, like the arteries, are supplied with vasa vasorum.

*The Lymphatic Vessels.*—The blood vessels are surrounded on every side by plasma spaces—irregularly shaped lacunæ, into which fluid transudes.

The lymph capillaries with a distinct endothelial wall unite as they pass upwards to form larger lymphatic vessels. The walls of these trunks are possessed, as in the case of the arteries and the veins, of three coats. The intima consists of a longitudinal fibrous layer, lined by a nucleated endothelium. The media is composed of circular unstriped muscular fibres with some elastic tissue. The adventitia consists of a loose fibrous tissue with a few longitudinally arranged unstriped muscular fibres. The walls of the lymph channels, therefore, as has been so clearly put by Hamilton, consist first of a homogeneous cement substance, then of an endothelium with elastic internal coat, later of endothelial and elastic tunica intima and tunica media, and lastly of vessels with three coats. The main trunks of the lymphatic channels are provided with valves which prevent the regurgitation of fluid, and at the mouth of the thoracic duct into the subclavian vein there is a large bicuspid valve which guards the opening.

## CHAPTER II.

### PHYSIOLOGICAL.

THE conditions of the blood flow, as regards the forces by which it is maintained, and the influences by which it is modified, must be fully grasped in order to approach the problems of disease with any prospect of successful study. It is, however, impossible in such a work as this to do more than cast a rapid glance at the general phenomena of the circulation, in order to ascertain the principles underlying the important functions which it subserves. It seems natural in considering the circulation to take up, in the first place, the phenomena presented by the heart. Not only is the development of its functions the most striking fact in the embryonic circulation, but in later life the indications which it gives are of the highest import as regards diagnosis and treatment. The action and reaction of the heart and vessels must be considered together as a whole, but the first place in a general survey of the physiology of the circulation must be accorded to the appearances connected with the heart.

#### THE HEART.

The essential function of the heart is to produce in the arterial system such a pressure as will cause the blood to flow continuously onwards and outwards. This is strictly analogous to what is termed "a head of water" in practical hydraulics. The object is attained by the vital process of contraction, through which the necessary force is pro-



duced, and the mechanical device of valves, by which the flow is directed. The heart is accordingly, as has so often been remarked, at once a suction pump and a force pump, but the fact cannot be too strongly insisted on that it is instinct with life, and brought into harmonious action with the other structures concerned in the circulation by the nervous system.

THE CARDIAC MOVEMENTS AND SOUNDS.—Under ordinary circumstances there is little obvious evidence of the pulsation of the heart; a slight impulse seen over the præcordia, and the movements of some of the superficial vessels, are the only appearances to be observed. To ascertain the course of events it is necessary to resort to experiment, in order to render the movements of the heart and great vessels visible.

On watching the exposed heart of any of the larger mammals, as has been done by every physiologist since the observations of Harvey, it is not difficult, after the eye has become accustomed to the rapid changes of form which it undergoes, to follow the sequence of movements presented by its different parts. The earliest of the cycle of events to be seen, after the long pause, is a slight flickering contraction of the superior vena cava, known to Haller and observed by Senac. A similar contraction can be observed in the inferior vena cava and the pulmonary veins, by specially exposing them, and it has been ascertained by the investigations of Colin, as well as of Brunton and Fayrer, that these movements continue after every part of the heart has ceased to contract. The quivering movement of the great veins is immediately followed by the systole of the auricles, seen most distinctly in that of the right. Its sudden swift contraction causes the sinus almost entirely to disappear, while the appendix, drawn backwards and to the right, becomes small and pale. A short pause ensues, and the heart remains to all seeming quite at rest; this brief period of repose is followed by two events—the relaxation of the auricles, and the beginning of the ventricular systole—which to the most careful scrutiny of the unaided eye seem perfectly simultaneous; it has, nevertheless, been proved that the auricles remain contracted until the ventricles commence their action. The right auricle becomes once more dark in colour as it resumes its former size and shape.



The apex of the heart tilts forwards and to the right, and the whole of the ventricular portion suddenly alters its form, so that instead of presenting a flattened curve it assumes a rounded outline; at the same time it loses its flaccidity and becomes hard to touch. The ventricles continue to contract, and while the long axis of the ventricular portion increases slightly in length, the transverse diameter is gradually reduced in size. It is quite clear that there are three distinct phases in the ventricular systole; in the first, the ventricles produce sufficient pressure to overcome that in the great arterial trunks, and in doing so they alter their form and become hard to touch; during the second, which is accompanied by a diminution in size, they expel their contents as the pressure upon them rises to a sufficient level; during the third, they remain for a brief time contracted after expulsion. In one of the larger mammals, a distinct expansion of the aorta and pulmonary artery can be seen along with the systole of the ventricles, and if the finger be applied to one of these vessels an impulse may be felt. The final phase of the cycle is a somewhat sudden return of the ventricles to their flaccid condition and flattened outline, and this is accompanied by an impulse, which may be seen and felt, in the great arterial trunks.

Two sounds accompany the movements of the heart. One of these, commonly known as the first, is lower in pitch and longer in duration than the other, usually termed the second sound. The first sound is followed by a short pause; the second by a longer interval. These sounds, known in some measure previously, but only understood during the present century, are still a source of controversy amongst physiologists. The first sound was correctly recognised by Laennec as occurring along with the ventricular systole, but the second, supposed by him to coincide with the systole of the auricles, was first placed by Turner with approximate accuracy at the end of the ventricular systole, and the fact was more thoroughly established by the researches of Hope and his coadjutors.

*External Movements.*—Attention must be bestowed upon the movements in somewhat greater detail. Since the obser-

vations of Ludwig it has been recognised that the long axis of the heart increases during the systole, while the size of the other diameters varies according to the extrinsic conditions under which the heart is placed during systole and diastole. If the heart of any mammal under observation is allowed to be in the thoracic cavity, it assumes during diastole a flattened form on account of the nature of its support, and during systole the transverse diameter diminishes while the antero-posterior increases. But if the heart is suspended in such a way as to rest upon no support during diastole, it has during that phase a more conical form; in the first stage of systole, according to Noël Paton, the antero-posterior diameter increases, and during the second stage both transverse and antero-posterior diameters diminish. Under all circumstances the long axis becomes greater during systole, as Ludwig found in a long series of observations.

The movements of the heart may be analysed by means of the graphic method. Since Marey applied the cardiograph to the investigation of the movements of the heart, many observers have devoted themselves to researches in the same direction, but unfortunately their results have been so far from harmonious that the interpretations of the tracings obtained have been nearly as numerous as the workers. Tracings may be obtained from the pulsation of the heart through the walls of the thorax, but much more may be learned from a study of curves from cases in which, on account of malformation of, or operation on, the walls of the chest, the movements of the heart have been easily accessible. The information, even when obtained under such favourable circumstances, has many limitations, and the results have been almost confined to the determination of the sequence and duration of the different phases of cardiac activity.

The character of the tracing depends entirely upon the part of the heart to which the cardiograph is applied—a point specially investigated by von Frey—and in the case of curves obtained from the pulsation of the heart through the walls of the chest, there is not only some difficulty as to the exact region of the heart giving the impulse, but also some uncertainty in regard to the nature of the intervening structures. The tracing is also modified by the amount of pres-

sure applied by the instrument employed, as Roy and Adami have illustrated by their observations. It must further be remembered that the details of the curve are conditioned by the nature of the instrument made use of in obtaining tracings. These considerations are sufficient to show that the form of the curve has large possibilities of variation, and since the publication of the first cardiogram by Marey, the tracings of those who, like Landois, Edgren, and Martius, have devoted special attention to the subject differ widely in many respects. It is not possible in this place even to refer to the points in which these curves vary.

The appearances presented by an ordinary cardiographic tracing will be discussed in the section devoted to the investigation of clinical phenomena. The cardiographic curve only gives the relation of the movements of the heart in regard to time and place; it throws no light on the strength or force of the cardiac contraction. Tracings obtained from the exposed hearts of the larger mammals present a general resemblance to those taken from the human heart under favourable conditions, and such curves have been of some real utility in showing the differences which exist between the movements of the several parts of the heart, even of neighbouring parts of the same ventricle, according to the site occupied by the instrument.

Several observers have been able to take tracings from the heart in cases of sternal or costal deficiency. In comparatively ancient times the famous case of Groux attracted much attention, and was the subject of study by almost every physiologist of eminence, but the opportunity presented by his extensive sternal fissure occurred before the graphic method was thoroughly established. An instance of sternal fissure in a healthy young man, whose case was fully described by Malet and myself some years ago, afforded an occasion for obtaining numerous cardiographic tracings. In this case there was a wide fissure in the upper part of the sternum extending as far as the fourth costal cartilages, and in the lower part of this opening the movements of the heart were remarkably prominent. There could be no doubt that the movements were caused by the *conus arteriosus*. Not only did the pulsation occupy the anatomical position of that part

of the heart, but the character of the movements seemed to bear out this conclusion. In the lower part of the hollow caused by the sternal deficiency, the pulsation showed the following character:—the state of rest was a prominent degree of fulness; the cycle commenced with a sudden increase of the swelling, followed by a swift wave-like subsidence downwards; this was in turn succeeded by a short sharp impulse, after which there was a rapid return of the fulness. On palpation the same sequence of events was ascertainable, the short sharp impulse being especially marked, and the sinking of the swelling gave to the finger a distinctly vermicular feeling passing from above downwards. Over the areas usually auscultated the cardiac sounds were quite normal. By means of the binaural stethoscope, which allowed of light pressure, the pulsating area was carefully auscultated. Accompanying the increased fulness, which began the cycle, was a faint blowing murmur, immediately followed by the first sound, which commenced with, and lasted until the end of, the downward subsidence, being accompanied by a soft low murmur; the short sharp impulse coincided with the second sound, which was very loud; the return of the swelling was attended by a scarcely audible blowing murmur.

With a direct cardiograph many tracings were obtained, and the movements were compared with the carotid pulse, as well as with the cardiac sounds. The apex beat unfortunately could only be felt when the patient was sitting or standing, and it was therefore useless for graphic purposes. Placing a finger upon the carotid artery, and watching the cardiograph when in motion, the arterial pulse appeared to be synchronous with the swift fall of the lever; still watching the instrument, and listening to the heart sounds with the binaural stethoscope, the commencement of the first sound was clearly simultaneous with the abrupt rise of the lever to its highest elevation, and it continued until it descended almost to its lowest level; the sharp impulse which followed was exactly coincident with the second sound. The tracing obtained by means of the cardiograph with the cylinder revolving at a slow rate gives the respiratory curve as well as that of the cardiac movements.



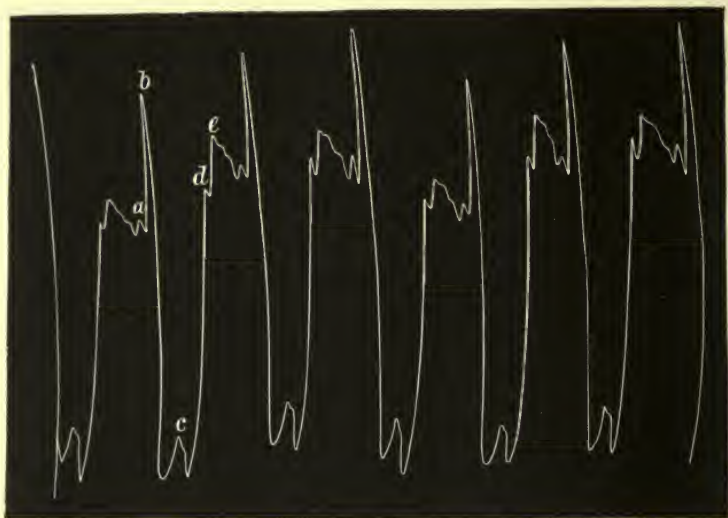


FIG. 14.—Tracing from conus arteriosus in sternal fissure.

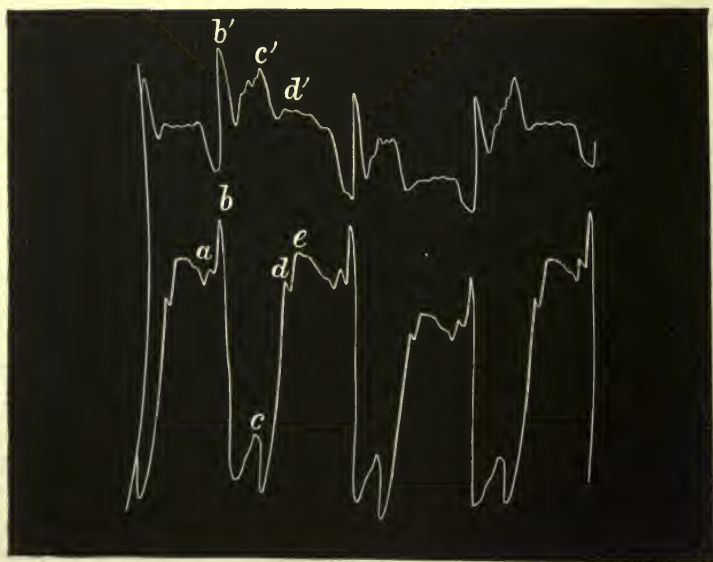


FIG. 15.—Simultaneous tracing from conus arteriosus and carotid artery.



By means of two levers the curves shown in Fig. 15 were obtained, in which the upper tracing is taken from the carotid artery, and the lower from the conus arteriosus.

In this figure it is clear that the arterial impulse coincides exactly with the sinking of the conus. The starting-point in the cycle of movements, as shown in Fig. 14, is *a*, which, from its relative rhythm, is caused by the auricular systole. The ascent of the curve which follows is due to the latent period of ventricular contraction, and it is followed by the sinking of the curve produced by the expulsive phase. The abrupt, but

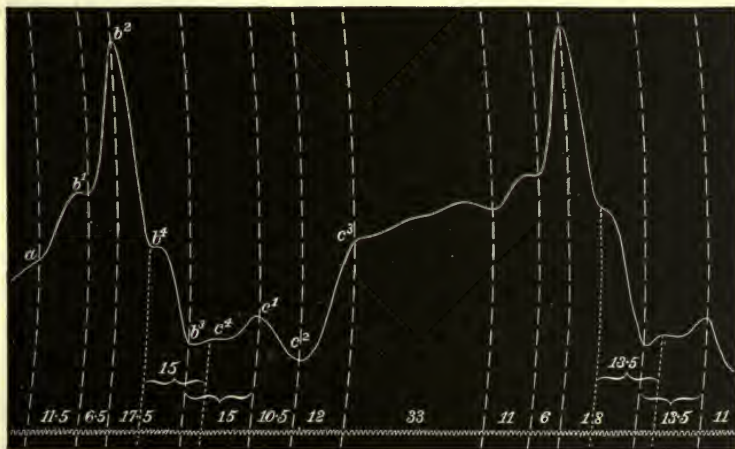


FIG. 16.—Tracing from conus arteriosus with curve of tuning-fork.

small, rise at *c*, coinciding as it does with the diastolic shock and second sound, and being synchronous with the katarotism of the arterial tracing *c¹*, marks the commencement of diastole. Succeeding this little rise, which so clearly marks the commencement of diastole, is the filling up of the heart by the blood entering during its relaxation, which causes the ascent of the line. Both figures read from left to right.

To analyse the movements more fully and to measure their duration further investigations were carried out by me; the cardiograph being employed with a rapidly revolving cylinder, on which were registered, along with the cardiac curve, the movements of a tuning-fork vibrating in hundredths

of a second. Part of one of the curves so obtained is shown in Fig. 16, which is to be read from left to right. It is needless to give a description of the curve shown by the tracing: it is only necessary to mention that  $a$  marks the beginning of auricular systole,  $b^1$  the commencement of ventricular systole—coinciding with the beginning of the first sound—and  $c^1$  the occurrence of the second sound. In the tracing given here, there is on the descending curve from  $b^2$  to  $b^3$ —which is caused by the emptying of the ventricles of the heart—a shoulder marked  $b^4$ ; and there is also, in advance of  $c^1$ , which marks the incidence of the second sound, an elevation  $c^4$ . On looking at the measured intervals of time recorded below the curve, an interesting fact will be noted. The period elapsing between  $b^3$  and  $c^1$  is precisely the same as that between  $b^4$  and  $c^4$ . The conclusion is obviously that  $c^1$  and  $c^4$  represent the diastolic recoil on the sigmoid valves, while  $b^3$  and  $b^4$  are the terminations of ventricular emptying on the two sides. Many other tracings taken from the same case show no shoulder in the position of  $b^4$ , and no elevation in the position of  $c^4$ , a point which will be referred to in a subsequent section. The measurements of the different phases, obtained by counting the number of complete excursions of the tuning-fork, are given in the figure to save trouble and facilitate comparison.

No. of Tracing and of Cycle.	Auricular Systole.	Ventricular Systole.	Diastole.	Entire Cycle.	No. of Tracing and of Cycle.	Auricular Systole.	Ventricular Systole.	Diastole.	Entire Cycle.
1. I.	·115	·345	·605	1·065	10. II.	·105	·390	·690	1·185
2. I.	·120	·350	·670	1·140	11. I.	·105	·365	·465	·935
3. I.	·125	·355	·670	1·155	II.	·100	·380	·495	·975
4. I.	·130	·375	·660	1·165	III.	·100	·355	·485	·940
5. I.	·125	·335	·455	·915	12. I.	·130	·385	·625	1·140
II.	·125	·345	·455	·925	II.	·105	·375	·615	1·095
6. I.	·125	·360	·490	·975	III.	·105	·355	·575	1·035
II.	·125	·355	·575	1·055	13. I.	·115	·390	·555	1·060
7. I.	·115	·375	·570	1·060	II.	·110	·375	·555	1·040
II.	·110	·360	·525	·995	III.	·110	·365	·470	·945
8. I.	·105	·375	·565	1·045	IV.	·110	·380	·465	·955
II.	·110	·380	·560	1·050	14. I.	·105	·390	·605	1·100
9. I.	·105	·335	·675	1·115	II.	·105	·390	·690	1·185
II.	·105	·325	·675	1·105	III.	·100	·380	·600	1·080
10. I.	·110	·395	·685	1·105	IV.	·115	·390	·625	1·140

In the preceding table are embodied the measurements of at least one complete revolution of every cardiogram obtained, taken on the basis of the second sound being the point of separation between systole and diastole.

The average absolute duration of each phase and that of the entire cycle are as follows:—

Auricular Systole.	Ventricular Systole.	Diastole.	Entire Cycle.
·112 sec.	·368 sec.	·578 sec.	1·057 sec.

The limits of absolute duration of each phase, as well as the difference, may be stated in this way:—

	Longest.	Shortest.	Difference.
Auricular Systole . . . . .	·130	·100	= ·030 sec.
Ventricular Systole . . . . .	·395	·325	= ·070 „
Diastole . . . . .	·690	·455	= ·235 „

and the difference, as compared with the shortest absolute duration, expressed in percentages, gives the following table:—

Auricular Systole . . . . .	·030 : ·100 = 30	per cent.
Ventricular Systole . . . . .	·070 : ·325 = 21·3	„
Diastole . . . . .	·335 : ·455 = 51·6	„

which clearly shows the extent to which the duration of each phase may vary from its lowest limit.

Leaving the absolute duration of the individual parts of the cardiac cycle, the variations in the relative duration of each, as compared with the entire revolution to which it belongs, may be arranged in the following percentages:—

	Shortest.		Longest.
Auric. Syst. in 14. II. is	8·8 per cent,	and in	5. I. is 13·7 per cent.
Ventric. Syst. „ 9. II. „	29·4 „	„	13. IV. „ 40·6 „
Diastole, „ 5. II. „	49·1 „	„	9. II. „ 61·1 „

All these tables show clearly that the most stable part of the cardiac revolution is the ventricular systole. The duration of the diastole—upon which the rate of the heart beat mainly depends—is the most variable, and next to it must be ranked the contraction of the auricles.

The measurement of the phases of the cardiac cycle has during modern times been the subject of careful investi-

gations by other observers besides myself. Volkmann attempted to estimate the duration of the movements, but the first approximation to accuracy was made by Donders. The latter observer, who accompanied the sounds of the heart by movements which were registered along with electric signals from a clock, found that the interval between the first and second sounds, which he took to represent the ventricular systole, varied between the limits of ·309 and ·327 sec., and was tolerably constant for the same individual with a high as well as with a low pulse-rate. In the sitting posture the systole, as compared with the whole cycle, was relatively shorter although absolutely longer. From such considerations Donders concluded that the systole has an independence of its own. By means of a Marey's sphygmograph, Landois carefully measured the different movements of the heart at the apex beat. To afford the means of easy comparison with my own results, the following table is taken from his paper:—

a. Dauer der Vorhofcontraction bis zum Beginn der Ventrikelsystole . . .	0,170	0,177
b. Dauer der Ventrikelcontraction . . .	0,155	0,192
c. Verharren des Ventrikels in der Contraction . . .	0,088	0,082
d. Vom Beginn der Diastole bis zum Schluss der Semilunarklappen . . .	0,066	0,072
e. Dauer der Diastole vom Schluss der Semi- lunarklappen bis zum Beginn der Pause . . .	0,259	0,200
f. Dauer der Herzpause . . .	0,393	0,407
Dauer des ganzen Herzschlages . . .	1,133	1,133

In this table *b*, *c*, and *d* represent the systole as measured by Donders.

As the result of a long series of observations Edgren gives the following average duration of the cardiac phases:—

Systole {	latent period . . .	·0934 sec.	} ·3276 sec.
	expulsion „ . . .	·0990 „	
	residual „ . . .	·1352 „	
Diastole {	commencing relaxation	·0520 „	} ·5348 „
	residual „	·4828 „	
Entire cycle for pulse-rate of 70 . . .		·8624 „	

The variability of the diastolic phase with different rates of pulse, which was so well brought out by my own results, has



been shown in the lower animals by Baxt, who caused a shortening of the cycle by stimulating the accelerating nerves. From his results it is clear that great changes in the diastolic phase are thus produced, while the systole is but little affected. The following measurements bear this out :—

Normal Heart.		Accelerated Heart.	
Systole.	Diastole.	Systole.	Diastole.
·253	·299	·210	·044

*Internal Movements.*—The changes which take place in the interior of the heart during its different phases are, on account of inherent difficulties, by no means absolutely known. By the anatomical method the condition of the walls and the position of the valves may be ascertained with considerable certainty; by experimental research, attempts have been made to investigate the movements of the valves, but the results are not yet beyond the possibility of misconception.

Anatomical researches into the position and relations of the different parts of the interior of the heart throw much light upon the mechanism by which the blood flow is carried on. Such investigations have been from time to time undertaken by different physiologists, and the matter has recently been the subject of an elaborate research by Noël Paton, in whose work will be found full references to the observations of previous writers. By an ingenious method of fixing the heart in the latent phase of contraction—before the opening of the arterial valves—and in the final phase of residual contraction—after the blood has been entirely expelled—he has been able to compare the position of the cavities and valves during ventricular systole and diastole. According to his observations, during the latent period the cavity of the right ventricle diminishes in its transverse diameter from drawing in of the external wall, while the antero-posterior increases on account of bulging forward of the anterior wall in the infundibular region. During this phase the septal cusp is closely applied to the septum, while the external cusp is pulled towards it, and the infundibular cusp is pressed closely against it from the action of the superior and anterior papillary muscles; the outer part of the auriculo-ventricular ring is at the same



time pulled downwards by the combined action of the papillary muscles, and of the muscular fibres surrounding the orifice. During this phase the cavity of the left ventricle is narrowed transversely and widened antero-posteriorly. The two cusps are at this time raised from the ventricular walls and drawn towards each other by the action of the papillary muscles. One important function of these muscles, and of the anterior mitral cusp, is to aid in keeping the aortic orifice open by their action on the membranous part of the auriculo-ventricular ring, as was in part appreciated by Onimus.

In the phase of residual contraction the cavity of the right ventricle is flattened and obliterated, except the conus arteriosus below the pulmonary cusps. The septal cusp is pressed against the septum, and the other segments are closely applied to it. The cavity of the left ventricle is entirely obliterated except a small cylindrical part below the aortic orifice. The two mitral cusps are applied flat against each other almost throughout their entire extent, only a small wedge-shaped space being left towards the auricles at the upper part of the valve. From these observations Noël Paton concludes that instead of the auriculo-ventricular valves being floated into a horizontal position, they are simply applied face to face, and close their respective orifices without any strain being placed upon them.

With regard to the arterial orifices, Noël Paton lays stress upon the fact that the anterior cusp of the aortic valve is placed upon the muscular cushion formed by the ventricular septum, originally described, as stated in the previous section, by Savory, so that this cusp, which, as Pettigrew showed, closes first and supports the other two, has an efficient buttress diminishing the stress it undergoes. A similar muscular support is found underneath the left posterior pulmonary cusp, upon which the other two cusps rest.

These results prove conclusively that much of the teaching contained in some of even the best of our systematic works on physiology as to the position of the valves is erroneous, and demonstrate the truth of the views advocated by Meckel, Burdach, Mayo, Reid, Pettigrew, Küss, and Marc Sée.

Attempts have been made to obtain tracings of the movements of the internal parts of the heart. Of such investiga-

tions the most important have been the researches of Roy and Adami, who obtained simultaneous tracings from the interior and the exterior of the heart. Their method was to introduce a hook into the cavities of the heart, which caught the cusp of the auriculo-ventricular valve, while the free end was attached by a string to a light lever. This traced the movements of the cusp on a revolving cylinder, which at the same time recorded the curves of the lever connected with the exterior of the heart. Roy and Adami came to the conclusion that the contraction of the papillary muscles was later in its commencement and shorter in its duration. There can be but little doubt of the fact that the papillary muscles are appreciably later in their contraction than the walls, the difference in time between the two events being, from the researches of Fenwick and Overend, about .05 seconds. But, as will be seen later, the inferences which Roy and Adami have drawn from their investigations are as yet unproved.

*Intra-cardiac Pressure.*—The variations of the intra-cardiac pressure have been investigated in two different ways:—by registering the curves obtained from the different cavities by the simple manometric methods of Chauveau and Marey, Fick, Fredericq, and Hürthle; or by the maximum and minimum manometer of Goltz and Gaule. According to the experiments of Chauveau and Marey on the horse, the pressure in the right auricle is 2.5, right ventricle 24.0, and left ventricle 128.0 mm. of mercury. The investigations of Goltz and Gaule showed that the maximum pressures might reach 20 mm. Hg. in the right auricle, and 62 mm. Hg. in the right ventricle.

With an exposed heart de Jager found that the right auricle could produce a negative pressure of from 2 to 6 mm. Hg., while in the corresponding ventricle there was a negative pressure of 5.38 mm. Hg. Such a negative pressure speaks for an aspiratory force attracting blood to the heart. It must further be borne in view that the pressure in the chest is, as was long since shown by Donders, negative. During an ordinary inspiration there is a negative pressure of about 11 mm. Hg., which rises during forced inspiration to about 70 mm. During the phase of expiration the negative pressure is about

3 mm. Hg., but forced expiration may give a positive pressure of 90 mm. Hg.

*Cause of the Movements.*—It would serve no purpose to recall the different views which have been held at various times as to the cause of the rhythmic movements of the heart, but a passing glance may be cast at those which have most materially contributed to the elucidation of the subject. The earliest theory of modern times is that of Haller, who believed the heart to be endowed with an inherent irritability, independent of all nervous influence, and excited to activity by the contact of the blood entering the cavities. This view was somewhat modified by Senac, whose opinion tended in the direction of allowing more influence to the nervous system.

We have seen that the movements of the heart begin with the great veins, pass thence to the auricles, and finally reach the ventricles. When the heart has been removed from the body of any animal the different parts of the heart die in the same order, and as the nervous ganglia are more numerous upon the terminations of the great veins and the adjacent sinuses than upon the rest of the auricles, and much more so than upon the ventricles, it used to be thought that they were the cause of the contractions of the heart. But the parts destitute of these ganglia may be caused to beat perfectly, as is stated by Foster, without in any appreciable degree differing from the ordinary movements.

From the facts in regard to the movements of the heart in the embryo, it must be clear that these movements are independent of nervous structures, and that the rhythmic contractions of the embryonic heart must be due to some inherent property of its protoplasm. The phenomena may fitly be compared to the periodic movements of many of the lower invertebrates. The rhythmic movements of many pelagic acalephæ, for example, are an expression of the same inherent property.

The results obtained by embryological observation and by experimental research lead therefore to the same conclusion, that the heart is endowed with an independent power of rhythmic pulsation—that it is, in short, automatic.

But there are one or two other points arising from a con-

sideration of the behaviour of the dying heart. It is easy to see that instead of being regular or rhythmic the pulsations of the heart become irregular or arrhythmic. Sometimes, as Fano has described, the movements fall into groups; a second and larger periodicity being imposed, as it were, upon the original rhythm. It is also not difficult to determine that the auricles and ventricles cease to preserve their normal relationship; the former often contract twice or thrice for one ventricular systole. The converse never happens—a ventricular contraction is always preceded by an auricular beat. As to the real cause of the contraction we know nothing. It is beyond all possibility of doubt that the pulsations are automatic; but so far we can only assume that they are produced by an inherent power of rhythmic activity in the ultimate muscle structure, and that the special characters of the cardiac muscular movements, as compared with those of the skeletal muscles, is due to the less differentiated condition of the heart-muscle.

*Propagation of Movements.*—With regard to the propagation of the pulsation from the auricles to the ventricles we are no longer in doubt, as the recent researches of His have cleared up the difficulties connected with the subject. Recognising that the cardiac contractions are automatic, and believing that the muscular fibres of the auricles are not continued into the ventricles, it has not been easy to understand how the wave of contraction could be propagated from the base to the apex. The observations to which reference has just been made, however, have shown that there is a continuous band of muscular tissue which extends from the posterior wall of the right auricle to the attachment of the aortic cusp of the mitral valve.

The contraction of the muscular fibres of the heart is characterised by some special features. When a stimulus has been applied to the muscular tissue the curve is marked by its long latent period, its gradual rise and its slow fall. In one important particular the cardiac muscle differs from ordinary muscle—the amount of contraction does not depend on the amount of the stimulus employed. The effects of stimuli moreover differ greatly according to the time when they are applied. Such considerations show that the muscular tissue of the heart is essentially different from that of the skeletal muscles.



*Cause of the Heart Sounds.*—The method of production of the second sound has been carefully studied by Ceradini, who employed an apparatus by means of which he could study the closure of the valves in the isolated conus arteriosus of the right ventricle. His conclusions are that the moment the blood pressure within the artery comes to equal that of the ventricle, the valvular segments fall together and initiate the second sound, which is only augmented, not originated, by any of the events of the diastole. His conclusions have been controverted by Sandborg and Worm Müller. Using the entire heart in an apparatus representing the circulation, they found that, at the instant the pressure below the semilunar valves is removed, and the pressure in the artery exceeds it, the blood flows towards the heart and initiates a movement ending with the closure of the semilunar valves. The closure of the valves is therefore produced by a backward movement of the blood in the lowest part of the artery, and is absolutely diastolic in time. Fredericq has carried out similar experiments with results of the same character. And Boyd has more recently investigated the subject, with the result of supporting the conclusions of Sandborg and Worm Müller, as well as of Fredericq, that the closure of the valves is diastolic.

The production of the first sound of the heart can scarcely yet be regarded as absolutely settled. Williams, in his address to the first Dublin meeting of the British Association, mentioned that it was produced by muscular action. His grounds for the statement were that in the heart of an ass, from which the blood had been removed so that the valves were in consequence unable to act, the first sound was still heard with each contraction of the ventricles. The results were corroborated by a committee consisting of Williams, Todd, and Clendinning, appointed to report on the subject. These observations were repeated with some modifications in different directions by Ludwig and Dogiel, and, in later years, by Yeo and Barrett. It must, therefore, be allowed that the heart in its contraction produces at least part of the first sound.

There can, nevertheless, be no doubt of the fact, which is generally admitted, that the first sound of the heart is not the same in the bloodless heart as in one acting normally. Haycraft,



who has, within recent years, devoted attention to this subject, points out that the first sound is far lower in pitch after the removal of the blood. The difference can only be that, in the normal heart, the tension of the auriculo-ventricular valves produces acoustic vibrations which are absent in those hearts from which the blood has been removed.

There are without doubt some difficulties in regard to the muscle sound, which is, according to all observers from Wollaston to Helmholtz, a low-pitched sound, having the period of a body vibrating not more than 40 times per second; whereas the first sound of the heart is, according to Haycraft, in the base clef, and has the pitch of a body vibrating between 100 and 200 times per second.

Helmholtz proved that the sound produced by the contraction of a skeletal muscle was almost entirely a mere resonance sound, the muscle itself undergoing disturbances at a period too low to be audible. Haycraft has pointed out that movements, due to want of co-ordination between the fasciculi within the skeletal muscle, take place, and he holds that these movements cause the membrana tympani of the observer to vibrate at its own period of 40 per second. He regards the muscle sound as a simple resonance sound.

*Intensity of Sounds.*—During recent times the intensity of the heart sounds has been carefully studied, more particularly by Vierordt.

The method followed in his investigations was by interposing, between the chest-wall and the ear, pieces of gutta percha, which conduct sound indifferently, and noting the number necessary for the disappearance of the sounds. The average results of his estimation in healthy people are given in the following table:—

*The Relative Intensity of the Heart Sounds.*

First sound :	4-10 Years.	11-20 Years.	21-40 Years.	41-50 Years.
The left ventricle .	751	758	768	637
The right „ .	491	577	602	516
Second sound :				
Aortic . . .	626	492	481	546
Pulmonary . .	778	660	568	539

It will be seen on comparing the figures for different ages that, during early years, the first sound on the left side of the heart is relatively much louder than that on the right. During later years the intensity of the sound generated at the tricuspid orifice increases, and the difference becomes less. With regard to the aortic and pulmonary orifices, it will be noticed that there is a progressive diminution in the intensity of the pulmonary second sound, and that, although the aortic second sound also becomes less distinct, it is, when compared with the pulmonary, relatively louder in later years, and at length absolutely exceeds it in intensity.

*Relation of Sounds to Movements.*—The relationship existing between the sounds and the movements of the heart has long been the subject of much controversy. The position of the first sound at the commencement of the systole of the ventricles is universally accepted, but it is far otherwise with the second sound. If we compare the work of Edgren, for example, with that of Martius, we find the greatest discrepancy in the position of the second sound in their tracings; Fredericq, who has carefully analysed their results, agrees with Edgren in placing it in the second part of the descending curve.

**ELECTROMOTIVE CHANGES IN CARDIAC ACTIVITY.**—The heart beat is accompanied by definite electromotive phenomena. These have been studied by several observers, amongst whom may be mentioned Engelmann, Marchand, and Burdon Sanderson and Page. Within recent times the whole subject has been thoroughly investigated by Waller. The electric variations of the heart may be investigated by means of Lippmann's capillary electrometer, which demonstrates the variations of potential associated with cardiac contraction. By leading off to the electrometer with a pair of electrodes (zinc, covered with chamois leather, and moistened with brine) strapped to the front and back of the chest, the mercury in the capillary tube may be seen to move with each beat of the heart. A record of the movements of the column of mercury may be obtained by photographing the oscillations on a travelling plate simultaneously with the movements of a cardiograph. Each contraction of the heart is accompanied by an electric variation. Analysis of such records shows that the electric variation precedes the

contraction of the heart by an average time difference of about .015 sec.

The electric variation of the human heart may be easily demonstrated by leading off by the two hands, or one hand and one foot, plunged into separate vessels of salt solution connected with the two sides of the electrometer. The column of mercury may be seen to move with each beat of the heart, but its movements are less than when the electrodes are strapped to the chest. By simultaneous records of the movements of the mercury and of the heart it may be seen that the former slightly precede the latter. On closer investigation of the phenomena it is found that on leading off to the electrometer from two points, one near the apex and the other near the base of the heart, there are several events accompanying the contraction. There is an initial phase preceding the systole, during which any point near the apex is negative to any point near the base. There is, further, a terminal phase preceding the diastole, during which the basal region is negative to the apical. According to the observations of Bayliss and Starling, the initial phase is itself double, and the phenomena are, therefore, triphasic. This, as Waller shows, is unquestionable proof that the variation is physiological, for there is no possibility of such an arrangement of altered contact at the chest wall.

THE FORCES WHICH FILL THE HEART IN DIASTOLE.—Several factors are concerned in filling the chambers of the heart during their diastolic phase. Amongst these may be noticed the negative pressure existing, under ordinary circumstances, in the thoracic cavity, first recognised by Donders. This negative pressure within the thoracic cavity necessarily tends, by a process of aspiration, to draw blood from the systemic veins towards the heart, and it must be regarded as one of the most important forces in filling the heart.

In addition to this there is, further, the negative pressure produced in its chambers by the active diastole of the heart. This negative pressure, first experimentally shown, as already mentioned, by Goltz and Gaule, gives, as the result of several experiments on the dog, in the left ventricle, from 100 to 320 mm. water, and in the right ventricle from 10 to 25 mm. water.

Similar experiments have been carried out by several other observers, of which those of de Jager seem to be the most important. With the chest wall open and the heart exposed—the aspiratory action of the lungs therefore entirely excluded—he found in the right ventricle a negative pressure of from 5 to 38 mm. mercury, and in the right auricle one of from 2 to 6 mm.

The result of these researches is to establish as an incontrovertible fact that during diastole the heart manifests an aspiratory action, and draws blood into its cavities. The explanation, however, of this force is beset with difficulty. Whether it be by the mere elasticity of the cardiac fibres, as was suggested by Magendie—an explanation apparently accepted by Goltz and Gaule, as well as by de Jager; or, as Spring suggested, that the longitudinal are constricted by the transverse muscular fibres during systole, and by their expansion during diastole bring about the dilatation; or whether, as Luciani urges, the muscular fibres are endowed with the faculty of actively lengthening themselves, cannot, in the present state of our knowledge, be definitely decided. One fact of the greatest importance must be mentioned here. Stefani found that the diastolic dilatation of the heart was more active while the vagi were left intact than when they were severed.

It is further to be remembered that the flow of the blood through the coronary arteries, which continues after the termination of systole, may have some influence in causing an active dilatation, and may, therefore, aid the aspiratory functions of the heart muscle. It seems probable that the general blood pressure and blood flow may be regarded as factors in filling the heart during diastole. But, inasmuch as the pressure and current in the venous system are small, they cannot be regarded as exerting much influence.

**THE NUTRITION OF THE HEART.**—One of the most important subjects connected with the causation of diseases of the muscular substance of the heart is connected with its nutrition. It is a well-known fact that, when a muscle is in action, the blood current through it is increased in rapidity, and it could hardly be expected that the heart would form an exception to this general rule. Certain observers, never-



theless, have held a contrary view. During last century, for instance, the opinion was expressed by Thebesius, that during the ventricular systole the semilunar valves closed the openings into the coronary arteries, and that it was, therefore, impossible for blood to enter them during that phase. Brücke revived this view in the middle of the present century, stating that no blood entered the coronary arteries during systole, and that the heart was flushed with blood during the diastole. Upon this view he founded the idea of an important regulating mechanism, holding that if blood entered the coronary arteries during systole the contraction of the ventricles would be hindered, while, on the other hand, if the blood entered these arteries during diastole its high pressure would favour the diastole. His opinion was opposed by Hyrtl, who, in the great majority of 117 autopsies, found the openings of both coronary arteries, or at least of one of them, to be higher than the free margin of the aortic cusps. Rüdinger observed that in the majority of cases the valves do not reach the opening of the coronary arteries. Brücke, on the other hand, stated that he, in 100 cases, only found four in which the coronary arteries were above the sinus of Valsalva. Ceradini and Krehl have confirmed the anatomical facts of Hyrtl, by showing experimentally that during systole the aortic cusps do not touch the arterial walls. There can be no doubt that in the overwhelming majority of cases the coronary arteries are above the level of the aortic cusps, and the valve, therefore, can in no sense interfere with the passage of blood into the coronary arteries during the systole of the heart.

That Brücke's views are erroneous has been decisively proved by the result of experiment. Not only did Hyrtl show that on cutting across a coronary artery in a living animal a jet of blood was thrown out in the beginning of the systole of the ventricles—a fact, however, which might possibly be open to a different interpretation as to the cause of the jet—but Chauveau and Rebatel have demonstrated that the pressure and the velocity of the blood increase in the coronary arteries with the commencement of ventricular systole. It was, however, observed that, while the blood



pressure remains high during the systole, the velocity falls to a very low level. This is probably caused by the very strong contraction of the heart which compresses the blood vessels, thereby sustaining or increasing the pressure, but interfering with the blood stream. At the end of the systole, without any increase of pressure, the flow in the coronary arteries is again increased, no doubt by the relaxation of the blood vessels. Their conclusions have been supported by the experiments of Martin and Sedgwick, who, by means of manometric investigations on dogs, found the blood pressure in the carotid and coronary arteries to increase simultaneously. The whole matter is a question of blood pressure and blood flow. As will be seen later, so long as the pressure is higher in the aorta than in the coronary arteries, the blood will certainly flow from the former into the latter. While Brücke, therefore, was in error as regards the filling of the coronary arteries during the systole, he was, to some extent, right with reference to the blood flow during diastole.

*State of the Coronary Arteries.*—The earliest experiments on the coronary circulation were made by Erichsen, who found weakening and stoppage of the heart on ligation of the coronary arteries. These were followed by the researches of Panum, who observed that when the coronary arteries were obstructed by substances that had been injected, there was a gradual slowing and cessation of the pulsation.

The size of the coronary arteries is the means of fixing the nutritive possibilities of the heart. It will be seen in a subsequent section that changes in the lumen of the coronary arteries, caused by disease, produce profound effects upon the myocardium. In this chapter the results of experiments upon these vessels must be considered. On clamping one of the coronary arteries in the rabbit, von Bezold observed that, after two or three minutes, the contractions of the left ventricle became irregular and peristaltic, and were followed by a perfectly arrhythmic flickering, which speedily ended in still-stand. The contraction of the right ventricle usually lasted somewhat longer than that of the left, but the appearances on the right side soon followed and resembled those of the left.

Similar appearances have been observed by Cohnheim and von Schultess-Rechberg in dogs. In their experiments the liberation of the coronary arteries never brought back the ordinary pulsations after fibrillary twitchings had once appeared. Indeed, after a short time the normal beat disappeared, even if the ligature were loosened before the appearances of fibrillary twitchings. Sée, Bochefontaine, and Bouzy produced similar effects, by injecting lycopodium powder into the coronary arteries. By von Bezold these results were regarded as the result of an interference with the nutrition of the heart muscle. As Tigerstedt, however, has shown, the blood supply to the ventricles may be stopped in the rabbit by firmly clamping the auricles. The aortic pressure falls to its minimum, and no blood flows into the coronary arteries, yet the clamp may be left for at least five minutes, and on its removal the heart resumes its normal pulsation. He concludes that it is through direct injury to the ventricular muscle that the pulsation is disturbed.

These observations do not throw any clear light upon the pathological changes to be afterwards described in connection with coronary lesions.

*Effects of the Condition of the Blood.*—The blood must have certain properties, in order to render it possible for the heart to perform its healthy functions. If it is deficient in the normal nutritive substances, or if it contains any toxic bodies, the cardiac energy must be impaired. It has been shown experimentally by Kronecker that, if the blood is replaced by a dilute solution of common salt, the pulse rapidly sinks so as to be imperceptible, while the heart, after exhibiting some feeble flickering movements, ceases to beat, and is unable to produce the least movement with the strongest stimuli. When oxygenated blood or serum is again given, the heart begins to manifest some feeble trembling movements, followed by a weak pulsation, which gradually increases until the beat is restored almost to the ordinary condition.

Stiénon has further investigated the effects of different conditions of the blood serum upon the heart beat. He observed that blood serum, after the removal of its sodium

carbonate by neutralisation by a fixed acid, slightly increased the activity of the heart, and that serum which had been boiled had an unfavourable effect, whilst fibrinoplastic substances were unable to restore its activity to an exhausted heart. He also sought to discover which of the substances contained in the serum must be present in order to give the heart the possibility of powerful and regular pulsation, and concluded that the presence of from  $\frac{1}{2}$  to 1 per cent of carbonate of sodium in a 6 per cent solution of common salt, in the presence of a soluble organic substance, probably of albuminous nature, provided the best possibilities.

Martius, from a series of observations, came to the conclusion that only those fluids containing serum albumin possessed the power of nourishing the heart, and he found that such substances as peptone, syntonin, egg-albumin, and milk casein possessed no powers of nutrition.

#### THE WORK DONE BY THE HEART.

Very different estimates have been formed as to the quantity of blood which leaves each ventricle during systole. A summary of these is given by Hoorweg. From 45 cubic centimetres, as estimated by Young, to 188, the figure stated by Volkmann, which form the extreme statements, there are a great many intermediate in amount. That which has been most commonly accepted is given by Vierordt, according to whom 180 c.c. is the average quantity ejected from each ventricle during systole. It is extremely probable that Vierordt's estimate is considerably in excess of the usual amount, and there can be but little doubt that the volume of blood leaving the ventricles varies considerably from time to time. As was stated previously, there is always a certain residuum of blood left in the valvular portion of each ventricle; it seems likely, moreover, that this quantity may, under certain circumstances, be largely increased. From the variability of the amount ejected during systole, it necessarily follows that any estimation of the work done by the heart must be merely approximate. Whatever may be the quantity

of blood leaving the heart, there can be no question of the fact that it must be equal on both sides of the heart. For, if either of the two ventricles ejected more than the other, there would at once be a disturbance of the balance between the systemic and pulmonary circuits.

The pressure in the aorta and pulmonary artery has to be overcome by the outflowing blood, and the amount of this pressure necessarily varies within wide limits. The pressure in the aorta may be taken as 150 mm. Hg., while in the pulmonary artery it amounts to about 60 mm. Hg. The force exerted and the work done by the heart may approximately be estimated by considering the pressure overcome. The left ventricle expels its contents against a resistance of about 150 mm. mercury, which must also be borne by the walls of the cavity. The force exerted must, therefore, exceed this resistance. In other words, as expressed by Tigerstedt, the force must be greater than would support a column of mercury with a base of 1 sq. cm., and a height of 150 mm. If the force be taken as equal to a column of 200 mm. the calculation is—

$$200 \text{ mm.} \times 100 \text{ sq. mm.} \times 13.6 \text{ gm.} = 272 \text{ gm.};$$

that is, the force is equal to the weight of 272 gm. resting on every sq. cm. As the pressure of the pulmonary artery is about  $\frac{2}{3}$  of that of the aorta, the force of the right ventricle may be estimated as 109 gm. for each sq. cm.

If the amount of blood ejected during each systole be 100 gm., and the aortic pressure 150 mm., or .150 m. Hg., it follows that

$$100 \text{ gm.} \times .150 \text{ m.} \times 13.6 = 204 \text{ gm. metres.}$$

But the velocity has also to be taken into consideration, and taking this as 0.5 m., with the standard 9.8 m. as the velocity of a falling body for one metre, and the formula  $\frac{pV^2}{2G}$ , we obtain as the law of efflux of fluid

$$\frac{100 \times 0.5 \text{ m.}^2}{2 \times 9.8 \text{ m.}} = 1.28 \text{ gm. m.}$$



The total is therefore

$$204 + 1.28 = 205.28 \text{ gm. m.}$$

The energy exerted by the muscular wall of the heart is in part changed into potential energy in the increased distension of the arteries, and in part into the kinetic energy represented by the momentum of the moving blood. The work done at each cardiac contraction may, as it is put by Starling, be calculated from the formula

$$W = BR \frac{BV^2}{2G},$$

where  $W$  stands for the work,  $B$  for the amount of blood expelled at each systole,  $R$  for the arterial pressure, and  $V$  for the velocity of the blood.

In this equation  $BR$  is the work done in overcoming resistance, and  $\frac{BV^2}{2G}$  is the energy employed in imparting the velocity to the blood. If we take the usually accepted figures of 100 gm. of blood as the amount expelled from the left ventricle at each contraction, half a metre per second as the velocity of the blood in the aorta during systole, and 150 mm. Hg. as the pressure in the aorta,  $BR = 204$  gm. metres of work, and  $\frac{BV^2}{2G} = 1.28$  gm. energy required to produce the velocity.

The velocity factor may therefore be neglected in considering pathological changes in the work thrown upon the heart. Any important increase in the work done by the heart can only be conditioned by an increase in one or both of the other two factors  $B$  and  $R$ , *i.e.*, the amount of blood expelled, and the resistance offered by arterial pressure.

#### THE ARTERIES.

It has been recognised since the work of Weber that the flow of any fluid in a system of tubes is different according as these tubes are unyielding and rigid, or distensible and elastic. With a continuous and uniform flow, the course of the fluid is practically similar in both kinds of



tube, but when, as is the case with the circulation of the blood, the fluid is driven in a series of rhythmic pulsations, the effect with the two forms of tube is entirely different. In the case of a tube composed of unyielding or rigid material, it escapes at the distal end in a series of intermittent jets; but when it is so driven into a yielding but elastic tube, it escapes in a continuous stream. The effect of elasticity is not only to convert an intermittent into a continuous flow, but also to relieve the force propelling the fluid by allowing, through expansion, only part to be driven on. An interesting effect of intermittence has been shown by some experiments of Hamel. This observer found that when a continuous stream was kept flowing through a frog's muscle, an œdematous condition was produced, and that when the stream was rendered intermittent the œdema disappeared.

The pressure of the blood, apart from variations produced by cardiac energy, arterial tone, and peripheral resistance, is subject to the ordinary laws of hydrostatics, and undergoes differences produced by gravitation. The pressure is equal in the same horizontal plane, and variations in hydrostatic pressure are due to differences of level. Following Thoma, the hydrostatic pressure of the blood may be spoken of as hæmostatic, while the pressure produced by the different factors of the circulatory mechanism may be termed hæmodynamic. Differences in hæmostatic pressure may be stated in terms of the difference of level of a column of blood, or, for greater convenience, of a column of mercury. As the specific gravity of blood is on an average about 1.05, while that of mercury is 13.59, the latter may be said to be approximately thirteen times heavier than the former. The hæmostatic pressure may be estimated from the point of exit at the heart, and this pressure may be termed  $p$ . In the erect posture, the hæmostatic pressure at the head, a distance of about 520 mm. will be equal to  $p - \frac{520}{13} = p - 40$  mm. Hg. On the other hand, the hæmostatic pressure at the foot, under similar conditions, may be expressed as  $p + \frac{1300}{13} = p + 100$  mm. Hg. The hæmostatic pressure is, to some extent,

counterbalanced by the vascular tone, and it is, therefore, to some extent, under the control of the nervous system. But there are special provisions to meet it in the structure of those parts of the body which are more specially influenced by it. In the veins of the leg, for example, there are more valves than in any other part of the body, while the walls of the vessels of the leg are considerably thicker than elsewhere. The actual pressure of the blood is the sum of the hæmostatic and hæmodynamic pressures. If the pressure of the blood leaving the heart be taken as 150 mm. Hg., the hæmostatic, hæmodynamic, and actual pressures may be expressed in the following table:—

	Static.	Dynamic.		Actual.	
		Vein.	Artery.	Vein.	Artery.
Level of Head	$p - 40$ mm. Hg.	+ 20	+ 100	- 20	+ 60
„ Heart	$p$ mm. Hg.	0	+ 150	0	+ 150
„ Foot	$p + 100$ mm. Hg.	+ 20	+ 100	+ 120	+ 200

THE FLOW IN THE ARTERIES.—Several factors influence the course of the blood along the arteries. The most important of these is the propulsive force of the heart, but, in addition to this, we have to consider the amount of blood which is ejected, the nature of the arterial walls, and the resistance in the periphery.

The sectional area of the vessels increases with distance from the heart, and towards the periphery of the vascular system there is in consequence larger storage for the blood. It follows from this that the course of the blood stream becomes slower with increased distance from the heart.

It has been shown that the flow of fluid differs in certain particulars according as it passes along unyielding or yielding tubes, and that the arterial walls are endowed with a very high degree of elasticity, but it is necessary to look into the facts regarding this elasticity a little more closely.

That the degree of elasticity brought into play is a variable quantity has been recognised since the observations of Wertheim, and it has further been recognised that the amount of elasticity

in operation becomes greater the higher the pressure. But this subject has, in more recent times, been carefully investigated by Roy, who observed that in healthy arteries the distension, from which recovery could take place, increased with greater pressure up to a certain limit, bearing a definite relationship to the total extensibility of the arteries, and that, when this limit was passed, the amount with still further increased pressure gradually became less. Zwaardemaker also observed that the elasticity of arteries showed its maximum with medium pressure. As the result of these investigations it follows that, with high arterial pressure, each increase in the amount of blood driven out of the ventricle must in a high degree increase the blood pressure.

The distensibility and elasticity of the arteries cannot be regarded as in any sense independent forces in the maintenance of the blood flow. They simply constitute a reservoir of energy and a means of regulation.

It has previously been stated that the smaller arteries and arterioles are endowed with a high degree of contractility, and the amount of contraction which is present in the peripheral arterioles has a most important effect upon the flow of blood in the arterial system. The causes which lead to such contraction of the arterioles will be discussed further on, but one important point must be brought forward in this place.

It has been shown by Roy and Adami, as well as by Johansson and Tigerstedt, by means of plethysmographic observations on the heart, that, when the arterioles are contracted to a certain but not too great degree, the heart drives out more blood than when the pressure is low. If, however, on the other hand, the contraction is still higher, the amount of blood driven out by each systole becomes smaller. It therefore follows that a medium pressure is that which is most favourable for the onward progress of the blood.

There can be no doubt that the resistance is also in part under the influence of metabolic changes in the tissues, and that when, from any reason, whether blood impurity or cellular inactivity, there is a diminished attraction of blood to the tissues, the resistance must increase. Another point of importance to remember is that, when from any reason the

absorption by the lymphatics is impaired, the peripheral resistance must rise.

THE BLOOD PRESSURE.—The conception of arterial pressure may be said to date from the experiment of Hales, who, by means of a long tube placed in communication with an artery, observed that the blood rose to a considerable height. No additional knowledge upon this subject was obtained until Poiseuille, pursuing a similar line of research, employed a U-tube filled with mercury, and in this way was able to estimate more exactly the amount of pressure in the artery. The simple instrument employed by him was afterwards improved by Ludwig, and, as the kymograph, is now part of the outfit of every physiological laboratory.

The blood pressure varies very considerably in animals. In the horse, for instance, it often reaches 300 mm. mercury, in the dog it is about 170, and in the rabbit about 100. In man it probably varies from 100 to 200, and the average pressure in the aorta is generally estimated as being about 150.

In different parts of the body the arterial pressure varies, and it may be stated as a general principle that it is somewhat less at a distance from the heart than near it. This, however, is only a relative truth, because, as Hürthle has in recent times demonstrated, the pressure in the femoral artery is often higher than in the carotid.

There are three main agents in keeping up the arterial pressure, connected with the heart, with the arteries, and with the blood itself.

The energy with which the heart contracts in systole has an important influence upon the blood pressure, as is well seen when the cardiac contractions are modified by interference with the vagus nerve. If the inhibitory influence of the vagus is removed, there is at once a considerable rise in the arterial pressure, and when the inhibitory influence of that nerve is brought into play, by stimulating the peripheral cut end, the blood pressure at once falls to a low level.

The resistance in the peripheral arterioles is a powerful factor in maintaining blood pressure.

The amount of blood in the arteries is also a most important agent, which is not only seen in morbid conditions,



but which has been demonstrated experimentally. Direct losses of blood, as shown by Tappeiner, cause a remarkable fall in the pressure. The use of dry food, by withdrawing blood from the general circulation into that of the digestive organs, also, according to Pawlow, produces a considerable fall, and, as shown by the same observer, the ingestion of a nutritive fluid, such as soup, produces a proportional rise in pressure. Free secretion from any of the glandular apparatus produces a fall of pressure.

THE VELOCITY OF THE BLOOD.—The velocity of the blood, or the amount passing through a given space in a given time, has been measured by a number of physiologists. It is found to vary greatly in different animals, as well as in different arteries of the same animal; it is also subject to much greater variations than the blood pressure. In the carotid artery of the dog, for instance, Vierordt observed that the velocity during systole reached 297 mm., while during diastole it only attained 215 mm. per sec.; and Chauveau, in the horse, found that in the carotid artery, during these two phases, the rate was respectively 520 and 150 mm. per sec.

The conditions of the blood in the arteries present two main facts for consideration. There is, in the first place, a rise of pressure during systole, which is propagated onwards as a form of wave motion. This wave motion is to be absolutely separated from the flow of the blood itself. There is, in the second place, the propulsion of the blood itself, as it streams from the heart outwards to the periphery.

The arterial pulse consists in a wave of increased pressure, travelling from the centre to the periphery of the arterial system. It is therefore to be regarded as an oscillation of blood pressure above and below a mean level, in consequence of which the blood always travels in one direction, that is, from the higher to the lower pressure level. The factors upon which the arterial pulsation depends are the amount of the blood, the energy of the heart, and the resistance of the blood vessels. The features of arterial pulsation depend entirely upon the relations to each other of these three factors. The energy produced by the heart, leaving out of account the loss sustained through friction, is expended in two different direc-



tions. The larger proportion, as has been previously shown, is transformed into the potential energy stored up in the walls of the distended arteries. The smaller proportion is manifested as the kinetic energy of the moving column of blood. The features of the arterial pulse will be described in the section devoted to the investigation of clinical appearances in health and disease.

In regard to the second consideration, its great result is the conversion of an intermittent jet into a continuous stream. During diastole, when the heart ceases its contraction, the swift wave of pressure and the slower flow of the blood tend to produce a negative pressure in the great arteries issuing from the heart, and this tendency causes a backward wave of pressure and, to some extent, a backward flow of blood.

#### THE CAPILLARIES.

The principal agency in propelling the blood through the capillaries is the high arterial pressure, but there can be no doubt that the flow through these vessels is aided by the attraction of the tissues in which they lie.

The flow of the blood in the capillaries is mainly characterised by being continuous, and by the fact that not merely transudation of plasma, but also the transmigration of corpuscles is freely allowed, under certain circumstances, through the walls.

The blood pressure in the capillaries has been estimated by several observers, more particularly by von Kries, and by Roy and Graham Brown. The principle upon which these investigations have been carried out is that of ascertaining what degree of pressure upon a region rich in capillaries is required to empty these vessels. As the result of such investigations, the capillary pressure in the frog has been estimated as from 7 to 11 mm. Hg. In man it is probably equal to between 20 and 30 mm. Hg.

The velocity of the current in the capillaries is estimated by observing, with the microscope, the passage of the corpuscles in the axis of the stream and measuring by means of the micrometer the time occupied in traversing a certain distance.

Such observations have been made by many inquirers, more particularly by Vierordt, who found that the rate of flow was from  $\cdot 5$  to  $\cdot 9$  of a millimeter per second.

One remarkable feature connected with the flow of the blood in the capillaries is that the coloured corpuscles keep the axis of the stream, while the colourless move along in its periphery. The most probable explanation of this appearance is that of Hamilton, who attributes the fact to a difference in the specific gravity of the corpuscles. Hamilton holds that the red blood corpuscles are of about the same density as the plasma, while the white blood corpuscles are of lighter specific gravity, and that they, therefore, tend to float upwards and to be displaced by those which are heavier.

The aim of the capillary circulation is to bathe every tissue in the body with blood, and it may be said that the purpose of this is twofold. The object of most of the capillaries, as for instance those of the muscles, is to supply fresh blood containing suitable nutriment, and, at the same time, to remove from the tissues impure substances, which are carried back into the general circulation. The obvious intention of other capillaries is mainly depurative. This is well seen, for instance, in the renal vessels, as almost the sole function which they perform is to remove from the blood impure substances.

### THE VEINS.

The flow of the blood in the veins is mainly produced by the high pressure in the arterial system, but after passing through the capillaries this force, primarily derived from the energy of the heart, is greatly reduced, and other agencies assist the current along the veins towards the heart.

The venous valves contribute to this end, because, directed forwards as they are, they prevent the possibility of a venous reflux, and, therefore, make provision for the onward passage of the blood when any lateral pressure is exerted upon the veins. Such lateral pressure comes from two main sources—muscular contraction by compressing the veins drives blood onwards, and, in the case of many veins which accompany arteries, the expansion of the arteries following the cardiac

systole produces a similar effect. But a large number of veins in the body have no valves. There are, however, other influences which assist the flow in such vessels. The negative pressure in the thoracic cavity, as already seen, has a powerful influence in drawing blood towards the heart, and the negative pressure produced by the cardiac diastole has the same effect.

The venous pressure is greater in the peripheral veins than in those which are nearer the heart. In the femoral vein of the sheep, for example, as observed by Jacobson, the blood pressure was 11·4, while in the brachial it was 4·1, in the facial 3·0, and in the subclavian, jugular, and innominate veins it was 0·1 mm. Hg. It is quite obvious that these degrees of pressure, as the veins approach the heart, must be caused by the aspiratory force within the thorax, as otherwise, with the diminishing area of the veins near the heart, there could not fail to be an increase of pressure.

A true venous pulse, by which is understood a perceptible pulsation in the veins from the periphery to the heart, is sometimes observed, and will be fully described in the chapter upon symptomatology. It is only necessary in this place to say that it is produced by the direct transmission through the capillaries of the oscillations of pressure in the arteries.

#### THE PULMONARY CIRCULATION.

The circulation of the blood through the lungs must be briefly noticed. It is influenced by most of the factors affecting the systemic circulation, but it is more affected by certain of these influences, while others are, for the pulmonary circulation, non-existent. Since the force exerted by the right ventricle is much less than that of the left, it follows that the blood pressure in the pulmonary circulation is much lower than in the systemic. The pressure in the pulmonary artery has been ascertained by several observers. Beutner, investigating the pulmonary pressure of the dog, found it from 28 to 31 mm. Hg.; Licht-heim found it from 10 to 33 mm. Hg., and Bradford and Dean observed a pressure of from 16 to 20 mm. Hg. As already mentioned, the amount of blood ejected by the right ventricle must be exactly equal to that driven out by the

left, otherwise the circulation would very speedily be brought to a standstill.

The respiratory movements of the thorax exercise not only great influence upon the pulmonary circulation, but produce distinct effects upon the systemic blood current. In ordinary inspiration there is in the air-passages a negative pressure of about 1 mm. Hg., while in ordinary expiration it becomes a positive pressure of about 2 mm. Hg., and these pressure variations influence the blood in all the vessels within the thorax.

These varying pressures within the thorax affect all the intra-thoracic organs of the circulation, but more especially act upon the auricles and veins, on account of the thinness of their walls. The elasticity of the lungs in their distended condition necessarily bears part of the atmospheric pressure. The tension of the lungs results in a negative pressure equal to 10 mm. Hg. during inspiration, and of 5 during expiration. If along with this pressure we consider the pressure within the respiratory passages, that is to say, the pressure of  $-1$  and  $+2$  mm. Hg. already mentioned, for inspiration and expiration, we get during inspiration a total negative pressure of 11, and during expiration of 3 mm. Hg. In other words, the pressure upon the heart and great vessels, taken as at sea level, may be expressed as equal to  $760 - 11 = 749$  mm. Hg. during inspiration, and  $760 - 3 = 757$  mm. Hg. during expiration. There is, in short, a difference of pressure equal to 8 mm. Hg. between inspiration and expiration.

These changes in pressure produce a variation in the flow of the blood, more particularly by their effects upon the right auricle and the venæ cavæ; during inspiration there is an increased flow towards the heart, and during expiration it is diminished. A larger amount of blood therefore must be allowed to flow into the aorta and pulmonary artery immediately after an inspiratory effort, and in these vessels there must in consequence be necessarily a higher pressure, while during expiration the opposite will take place. With forced respiratory efforts the varying intra-thoracic pressures become much more marked, since it is well-known that during forced inspiration the negative pressure may amount to 70 mm. Hg., while during forced expiration the positive pressure may be



equal to 90 mm. Hg. Respiration of rarefied air leads to an increased blood pressure, and breathing compressed air reduces it.

The systole and diastole of the heart affect the air pressure in the respiratory tubes, and the fluctuations of pressure in the contained air are apparently produced by the variations in the size of the heart. Curves have been obtained by Landois and Haycraft and Edie, which show diminution of pressure during cardiac systole, and increase during diastole. As is well known, an analogous phenomenon is met with on auscultating the lungs. In many instances, either on account of a lack of elasticity in them, or possibly an increased size of the heart, the respiratory murmur is interrupted or jerky, in consequence of the cardiac movements.

During inspiration there is a widening of the pulmonary capillaries, so that more blood is drawn into the lungs. During expiration there is an opposite effect, and from the greater pressure during this phase blood must be expelled from the lungs. It has been shown by Spehl that during natural inspiration the lungs contain from  $\frac{1}{13}$  to  $\frac{1}{12}$  of the entire blood in the body, while during ordinary expiration they only contain from  $\frac{1}{18}$  to  $\frac{1}{15}$  of it.

#### THE DURATION OF THE BLOOD CIRCUIT.

The length of time occupied by any given particle of blood in completing the entire circuit of the systemic, as well as the pulmonary circuit, has been often measured. The first attempt in this direction was made by Hering, and since the date of his researches all other observers have employed similar methods to those which he used. The essential feature of his method was the injection of some innocuous, but easily recognised, substance into a vein, and the examination at short intervals of blood from another vein in order to see how soon the injected material would appear. Hering's observations were made upon horses, and from them he concluded that the time occupied in completing a circuit from the jugular vein of one side, to the corresponding vein of the other, was about 26.2 seconds. Vierordt somewhat improved



the details of the method employed by Hering, and from many investigations came to certain definite conclusions. He found that the average duration of a complete circuit of the blood differs considerably in different animals, and that it is always shorter in those which are smaller. He further observed that the number of pulsations necessary to drive the entire quantity of blood once through the body in all animals, independent of their size, is almost equally great, varying as a rule from 26 to 29. This is shown in the following table:—

Animal.	Duration in seconds.	Pulse-rate per minute.	Number of pulsations.
Rabbit . . .	7.46	210	26.1
Goat . . .	14.14	110	26.0
Dog . . .	16.7	96	26.7

Vierordt further found that the average duration of the blood circuit in different animals is related to the average duration of an entire cardiac cycle, so that the quantity of blood which flows in a given time through a given weight of body, does so the more quickly, the smaller the animal. For example, the quantity is for the rabbit 592 gm. per kgm. per minute, in the goat 311, in the dog 272, and in the horse 152. In the same way the amount of blood leaving the heart is greater in relation to the body weight in smaller than in larger animals. The following table shows this:—

Animal.	Blood gm.	Per minute proportion.	Body kgm.	Weight proportion.
Rabbit . .	812	10	1.37	10
Goat . .	1,166	14	3.75	27
Dog . .	2,504	31	9.2	67
Horse . .	58,800	720	380.0	2,770

Inasmuch as the blood pressure in different animals varies comparatively little, it follows that the work of the heart in relation to the size of the body is greater in small than in large animals.

### THE LYMPH CIRCULATION.

The lymph which bathes the tissues is simply a transudation from the ultimate ramifications of the blood vessels, and is present in such an amount as suffices to render the metabolic

processes possible. In health there is no tendency to any accumulation or excess. The lymph differs from blood plasma in the proportion of its constituents. It contains less albumin and more water, while there are also the products of tissue change. Its specific gravity is from 1012 to 1022. It has an alkaline reaction, and a very feeble power of coagulating spontaneously. The amount of lymph which is present depends on the relative amount of transudation and resorption, on the balance between afflux and efflux.

The transudation of the fluid is manifestly connected with the blood pressure and varies directly as it is increased or diminished. The resorption depends on the natural circulation of the lymph. This apparently is partly due to the pressure in the plasma spaces derived from the blood pressure. The circumstance that the pressure within the large veins, into which the main lymphatic vessels open, is less than that in the tissues, which in turn is less than that within the small arteries and capillaries, is also an important factor. It must also be borne in mind that the movements of respiration have an aspiratory influence upon the lymph channels as well as upon the great venous trunks, that the lymph vessels have contractile walls, and further, that the contraction of the muscles must favour the return of lymph.

#### THE INNERVATION OF THE HEART AND BLOOD VESSELS.

Although, as has been fully insisted upon, the pulsations of the heart are absolutely automatic, and the vessels are possessed of elasticity, so that, as a mere piece of mechanism, the heart and blood vessels are perfectly able to sustain the circulation, the apparatus would, notwithstanding, speedily cease to work smoothly, were it not in some way regulated. This regulation is effected by means of the nervous system.

In all mammals—in fact, in most vertebrates—when the vagus is divided in the neck there is usually an increased frequency of the pulsations of the heart. This effect is by no means constant; in many instances no change is produced on section of the nerve, but if the lower end of the cut nerve be stimulated by electricity there is weakening, slowing, or arrest

of the pulsations, according to the strength of current which has been employed. On the other hand, if the sympathetic in the neck be cut, no effect may be produced; if, however, it be stimulated, the beats of the heart become increased in strength and frequency. The primary effects produced by stimulation of the vagus are followed by a period of greater activity after the interval of rest, while the converse takes place in the case of the sympathetic. The vagus nerve, therefore, is a channel by means of which inhibitory influences are carried to the heart, while the sympathetic nerve may be regarded as the means by which augmentor impulses are conveyed to it. Such influences may be conveyed by reflex action as well as directly.

The familiar illustration afforded by the ear of the white rabbit furnishes an excellent example of the nervous control of the circulation. When the ear is attentively observed it may be seen to undergo alternate redness and pallor. In the former condition all the blood vessels are seen to become wider, in the latter they are observed to contract. They, for the most part, however, maintain an intermediate condition which is that commonly known as vascular tone. If, now, in such a rabbit under chloroform, the sympathetic nerve be divided in the neck, the ear becomes redder and warmer, losing, from the time of the operation, the rhythmic movements of contraction and dilatation of the blood vessels. On stimulating the upper end of the cut sympathetic, the ear becomes pale, and the blood vessels are seen to have shrunk to their smallest possible limits. But on ceasing to stimulate the upper end of the sympathetic the ear at once becomes flushed and warm again. These facts prove that influences commonly called vaso-constrictor pass upwards by the sympathetic to the ear.

Another familiar illustration is furnished by the sub-maxillary gland. If the chorda tympani in its course to it be divided, but little effect is produced, but if the cut end of the nerve nearest to the gland be stimulated, it becomes enormously enlarged, its vessels undergo great distension, and a direct venous pulse may be seen beyond it.

These observations prove that influences of a vaso-dilator character are carried by the chorda tympani to the gland.

The nerves connected with the circulation naturally fall

into two groups, those which are connected with the heart, and those which are connected with the blood vessels. The course of the nerves passing to the heart has already been fully described. The cardiac nerves include inhibitory and augmentor fibres passing to the heart and depressor fibres coming from the heart.

The inhibitory fibres pass down to the heart in the trunk of the vagus nerve. They do not, however, belong to the vagus, but start from the spinal cord by means of the spinal accessory nerve. It has been shown that if the roots of this nerve are divided, while the vagus nerve is uninjured, stimulation of the vagus trunk fails of its usual effects. The augmentor fibres have their origin in the anterior roots of the second and third dorsal nerves, and, reaching the sympathetic system by the white rami communicantes, they pass through the first dorsal or stellate ganglion, through the annulus of Vieussens and the inferior cervical ganglion, whence they reach the heart usually through the lower cardiac nerves.

In some animals, *e.g.* the rabbit, the depressor fibres pass upwards in a separate definite nerve, which can be anatomically isolated, join the superior laryngeal branch of the vagus, and thus reach the bulb. In human anatomy, the depressor fibres pass upwards by the cardiac nerves to the superior laryngeal and vagus trunk, whence they pass to the bulbar centre.

There are some differences in structure in these fibres. Gaskell has shown that the inhibitory fibres are medullated throughout their course in the spinal accessory, vagus, and cardiac nerves; and that they probably remain medullated in their entire course. The augmentor fibres are medullated in the anterior roots, communicating branches, and sympathetic channels as far as the stellate and inferior cervical ganglia; they are, however, from this point outwards non-medullated.

The vaso-constrictor nerves have their origin in the spinal cord. They leave the spinal cord by the anterior roots of the dorsal nerves and pass to the sympathetic system by white communicating branches. The branches from the upper dorsal nerves reach the cervical ganglia and pass upwards, furnishing also branches to the arms through fibres passing from the stellate ganglion to the brachial plexus. The lower dorsal

nerves give branches which join the splanchnic nerves of the abdomen, and furnish nerves going to the lower limbs.

The vaso-dilator fibres have not been so far thoroughly traced. The only fact as to their origin is that, like the vaso-constrictor nerves, they emerge from the spinal cord by the anterior nerve roots.

Gaskell has pointed out that the two kinds of vaso-motor nerves are distinguishable from each other. Both are of the small medullated kind, and both leave the cord by the anterior nerve roots; but the vaso-constrictor nerves pass from the spinal nerves to the sympathetic system by white communicating branches, and return to the mixed nerve by gray communicating branches; while the vaso-dilator nerves accompany the spinal nerves and remain medullated until they reach their final visceral ganglia.

From an anatomical point of view the cardiac and arterial nerves fall into two groups. Vaso-constrictor and cardio-augmentor constitute a well-defined class; while vaso-dilator and cardio-inhibitory constitute another. The nerves belonging to the first class are non-medullated and excite muscular action; the nerves of the second group are medullated and restrain muscular contraction.



## CHAPTER III.

### PATHOLOGICAL.

THE special details of morbid changes in structure and function do not present themselves for consideration in this chapter—they will naturally arise in connection with the individual diseases to be described afterwards; the general principles connected with disturbances of the circulation must form the sole subject of the present part of the work. In order to render this short sketch in some degree comprehensive it is necessary to consider in a concise manner some general facts regarding the causation, the production, and the results of circulatory disturbances.

#### ETIOLOGY.

Considerations in regard to the general etiology of circulatory disturbances can alone be allowed a place in this connection. The special causes of particular diseases will be discussed under their proper heads, but some facts of a more general character may be grouped together in this place. Such etiological factors fall naturally into two groups. Certain influences belong to the individual and may therefore be regarded in the light of personal or intrinsic causes, others are exerted from without and may in this way be looked upon as impersonal or extrinsic causes.

**INTRINSIC CAUSES.**—Heredity plays a considerable part in the evolution of cardiac disease. Instances illustrative of this fact will be adduced in the sequel, and some of the most

important observations of previous writers will be referred to. Heart disease is present in certain cases at birth, sometimes in the form of congenital malformations, sometimes in the form of more restricted lesions which are not usually classed under this head. In many other instances, moreover, cardiac lesions develop in after life in the children of those who have been victims to similar affections, and it must be granted that the descendants of such persons show a tendency to the evolution of the diseases in question.

Age is an important member of this group. Certain diseases which have a tendency to leave a legacy of circulatory disorders are most frequently found in early life. Of this group rheumatism must be cited as the most frequent cause. It follows that those types of circulatory disease most likely to have their origin in this affection will be found during the early periods of life. Pericarditis and endocarditis in their acute forms are accordingly diseases essentially of youth. A further deduction may also justly be made, that those valvular lesions most likely to take their origin in acute endocarditis will also be found more particularly in youth, and hence mitral disease, for example, is most commonly found in early years. To more advanced periods of life belong the degenerative affections of the heart and blood vessels, such as arterio-sclerosis, which, linked on the one hand with cirrhosis of the kidney, and on the other with dilatation of the aorta and hypertrophy of the left ventricle, may be taken as a type of disease essentially belonging to advanced periods of life. This particular affection may justly be regarded in the light of a general disease, in which the circulatory system undergoes the greatest local disturbance. Along with, and in great part resulting from, such degenerative processes, are aneurysms connected with the great arteries, and local dilatations of the heart, to which the term cardiac aneurysm is commonly applied. From one or other of these conditions may arise rupture with all its dramatic phenomena. None of these are at all common before middle life, although some of them do occur occasionally in comparatively early years. Age may even be held to have some degree of influence as regards the incidence of certain functional disturbances. Palpitation of dynamic origin, for

instance, is much more frequently found in early years than in the more advanced periods of life. On the other hand, many disturbances generally considered to be functional, such as interferences with the rate and rhythm of the heart, along with the graver forms of cardiac pain, are rarely found before middle life. These, however, are for the most part associated with morbid structural changes.

Sex is a factor of some importance, although it is extremely difficult to explain many points in connection with its influence. It is an undoubted fact that mitral disease is more common in women than in men, while aortic disease presents a converse relationship. It is not difficult to explain why aortic disease is more common in men than in women, seeing that it arises undoubtedly in many cases from long-continued physical strain, to which men are necessarily more subject than women, but the reason why mitral disease should be more frequent in women than in men is, so far as can be seen at present, inexplicable.

Occupation has a very powerful effect upon the condition of the circulatory organs. Incessant demands upon the activity of the heart produce results which vary with its nutritive possibilities. When there is an adequate supply of nourishment the muscular wall of the heart increases in size on account of hypertrophy, but if the nourishment be inadequate the muscular wall undergoes dilatation. Long-continued stress upon the blood vessels gives rise to loss of elasticity, and compensatory sclerosis, along with degenerations of the endocardium and arterial intima. Indolence and self-indulgence give rise to fatty infiltration of the heart, probably arising in part from excessive nutritive material circulating in the blood, but in some measure also from diminution of the metabolic processes. Personal habits in the use of certain articles of luxury, and in excesses of various kinds, play an important rôle in the evolution of cardiac and vascular affections, as will be seen in detail in the sequel.

**EXTRINSIC CAUSES.**—The existence of previous disease exerts the most powerful of all influences upon the state of the circulation. Amongst previous diseases most potent in this way are some of those which we are fain to regard as consti-

tutional; that is to say, diseases which in our present state of knowledge may be considered as having their origin in causes arising within the body.

Rheumatism may be allowed to hold the most important position in this group. Whatever the poison of acute rheumatism may be, it certainly has a remarkable affinity for the serous membranes, both endocardial and pericardial. This, however, is not all, because even in perfectly latent forms rheumatism plays also another important part in the evolution of cardiac disease. It is extremely probable that many of the instances of hereditary heart disease are due to the presence of the rheumatic poison. It is held by many that rheumatism is probably produced by a specific organism. This may in the future be proved, yet it may at present be provisionally allowed that a chemical poison is adequate to explain the phenomena. Other disorders, such as renal disease, give rise to cardiac affections resembling those produced by rheumatism, no doubt by means of the retention of faulty chemical substances. Moreover, as will be shown immediately, the action of organisms is a chemical process.

Gout is less important as a cause of cardiac disease. It nevertheless gives rise to affections both of the external and internal serous membranes of the heart. It is far more potent, however, as an agent in the production of degenerative lesions of the blood vessels. By its effect upon the peripheral arteries it gives rise on the one hand to increased pressure followed by cardiac hypertrophy, while, on the other hand, by its influence on the coronary arteries, it brings about various degenerative changes in the heart.

Diabetes, scorbutus, and purpura, as well as other constitutional affections, give rise to certain definite cardiac and vascular disorders, which will be dealt with in subsequent portions of this work.

Of the members of the large group of diseases which owe their causation to influences acting upon the system from without there are many which more or less frequently induce circulatory diseases. Among these are, in the first place, the acute infectious diseases, many of which, as scarlet fever, have malign effects upon the pericardium and endocardium. The



pyrexia which forms the most important symptom of these diseases has, moreover, as is also the case in rheumatism, a powerful effect upon the myocardium, even in those who recover. This is probably due to cloudy or hyaline changes, which may in minor degrees temporarily impair, if they do not permanently disable the muscular fibres.

Bacteriology has supplied the link which was wanting in order to render the connection between these general diseases and the circulatory organs clear. The presence of micro-organisms has been definitely established in most, if not all, of the acute and subacute diseases of the heart. In many instances the introduction of these organisms seems to be the sole cause of the disease, as, for example, in the case of septic endocarditis and septic pericarditis. In other cases, however, it cannot be doubted that double infection is present, as for example in many cases of acute rheumatism attended by endocarditis, in which the presence of various organisms can be detected upon the diseased valves.

The manner in which such organisms act has caused a considerable amount of discussion. It now seems clear that they produce their effects by chemical irritation. Buchner has shown that dead bacteria, or even the protoplasm of which they are composed, will set up changes in the tissues at least as thoroughly as the living organisms. The process is one of positive chemiotaxis with determination of leucocytes to the affected part. The chemical processes set up have a further influence in the prevention of coagulation. To these two methods of action must be added a third, that is destruction of tissue. Under the chemical influences induced by bacterial action the tissues are dissolved and disintegrated. These effects may possibly be due to a peptonising action of the bacteria or their products. These three different factors are associated with the tissue reaction which attempts to curb the influences at work by means of cell proliferation and new formation.

On account of the intimate connection which exists between the circulation and some of the most important organs of the body, certain local disturbances exert much influence upon the circulatory mechanism. Two instances



of this fact may be cited in this connection, albeit they are but examples of what may be found in many other parts of the body. Disturbances of the circulation within the lungs produce great effects upon the right side of the heart in consequence of the obstacle to the passage of the blood, while changes in the kidneys, by acting in a similar way, powerfully influence the left side of the heart.

Some of the more chronic processes produced by external agencies are at times concerned in the production of cardiac and vascular lesions. Tubercle and syphilis may be referred to in this connection. Both have affinities for the blood vessels, more particularly in certain regions such as the brain. Both manifest also at times a tendency to invade the central mechanism of the circulation. In the invasion of the system by malignant new formations the heart is sometimes attacked. Whatever may be the laws bearing upon this subject, they are at present quite obscure.

#### PROCESSES.

The various morbid changes which take place in lesions of the heart and vessels must have some consideration from the standpoint of General Pathology, since a brief reference to the processes of disease met with in connection with the circulation will render it more easy to appreciate the nature of the lesions to be afterwards discussed.

DEGENERATIVE CHANGES.—The heart, like other muscular substances, undergoes that simple form of degeneration commonly known as *cloudy swelling*. In the course of most infective fevers, and also in some local diseases, such as acute Bright's disease, that have a powerful effect upon the general system, the changes summed up under this term are found. They are probably the result of the action of toxic substances which modify the protoplasm. The heart is somewhat enlarged, and has a pale cloudy appearance on section. The appearances observed are a swelling of the cells or fibres which become granular and cloudy, while the nuclei become less distinct.

Changes in the distribution of fat occur in very varied conditions, and the effects of *fatty changes* vary with the different methods in which the tissues are invaded. There may be, in

the first place, an accumulation of fat in those parts which normally contain it, as, for instance, the epicardium. Such deposits may be temporary or permanent. Whether the accumulation is due to excessive supply of fat-producing substances, or to faulty removal, it is impossible to say; probably both of these processes are effective in varying degrees. If the deposit is large or long continued, it necessarily impairs the functions of the heart, and is apt to lead to further changes. There may, in the second place, be a deposit of fat in tissues which are normally free of it, as, for example, in the inter-muscular tissues of the heart. In the fatty heart, which has just been referred to, there is a great tendency on the part of the epicardial fat to spread between the muscle fibres, more particularly of the right ventricle, and the process may extend as far as the connective tissue underlying the endocardium. These two forms of fatty deposit are usually included under the term fatty infiltration. It is not easy to state the causes of such accumulations of fat, but it must be obvious that there are hereditary tendencies in this direction, while certain habits, including faulty diet and deficient exercise, possess considerable influence. There is, in the third place, fatty degeneration. This differs from the two forms of fatty infiltration in the important particular that the fat is formed at the expense of the protoplasm of the cell. This change may follow in the wake of fatty accumulation and infiltration, but it is produced by a number of processes causing serious disturbance of the nutrition. Amongst the more important of these are (*a*) changes in the blood, as in various forms of cachexia and inanition; (*b*) lessened nutritive value of the blood, as seen after profuse hæmorrhages and in pernicious anæmia; (*c*) interference with the vitality of the tissues, as in pyrexia and venous engorgement; and (*d*) toxic conditions, whether arising from organisms as in diphtheria, or chemical substances as in alcoholism. The appearance of the heart differs somewhat according as the process is general or localised. When it is general, the entire organ appears paler and softer than in health; when localised, there are patches of paler colour alternating with others of normal hue, as in the tabby heart of pernicious anæmia. The cellular substance is obscured,

and contains fatty globules which may entirely obscure the nature of the tissue and the nucleus of the cell. The protoplasm is destroyed, and on dissolving the fat out of the tissue the cell appears vacuolated.

Occurring, so far as is known, as the result of certain coagulative processes, *hyaline degeneration* is usually observed in acute diseases. The muscle substance apparently absorbs some material which has the power of producing coagulation. In certain cases the tissue breaks up into hyaline masses from this coagulative change. This is more especially the case in some of the infective diseases, such as enteric fever and diphtheria. The fibres are, as a rule, broader than the unchanged fibres. The organ on naked eye examination has a somewhat cloudy appearance.

Under ordinary circumstances, as has already been mentioned, the heart goes on increasing in size during the entire lifetime of the individual, notwithstanding the loss of bulk undergone by other tissues during advanced years. In many cases of phthisis, and in the course of the cachexia of malignant disease, the heart undergoes *atrophy*. This must obviously be produced by a diminution in the size of the cellular elements, or by a diminution in their number; probably in most instances there is both diminution in size and decrease in numbers. These changes must be the result of loss of balance between decay and regeneration. There is a physiological limit to the repair of waste, and therefore, when the cells reach the senile stage, atrophy becomes evident. In many such instances there is simple atrophy without degeneration. Having played its part a cell disappears from the stage, and its place is not refilled. This is an entirely different process from the wasting produced by degeneration, in which the cells undergo some of the changes which have been referred to, and are either removed or replaced by lower tissues.

Deposits of calcium, either in the form of carbonates or phosphates, are often found and are usually associated with some of the salts of magnesium. This process is termed *calcareous infiltration*. It may occur in senile tissues, especially the vessel-walls—more particularly the intima or the media—in which case infiltration is generally preceded by fibrosis

or atheroma. In such a case there must be a previous weakening of the circulation of the part which favours the deposition of lime salts. It is also to be seen in the connective tissue which owes its origin to chronic irritation, as, for example, in the newly-formed fibrous tissue of the pericardium in chronic pericarditis. The nature of the process which leads to the deposition of lime salts is absolutely unknown. Some salts of lime are always present in the blood and lymph, and no more definite hypothesis can be assumed at present than that soluble become altered into insoluble salts.

The well-known change usually termed *pigmentary atrophy* is essentially a senile process. With advancing years there is a tendency to the deposition of pigment in several organs, one of which is the heart, but the causation of this process is unknown. In certain localities, such as the liver, the pigment is ferruginous and may therefore be directly obtained from the blood, but the pigment which is deposited in the muscular fibres of the heart is free of iron. It is collected chiefly around the nuclei, in which position it obscures the striation of the cells. It is by no means always confined to old age, and appears as a consequence of cachexia or marasmus. When so excessive as to give rise to a brown discolouration of the heart, it is termed brown atrophy.

REACTION PROCESSES.—Certain acute processes must be considered, inasmuch as these changes are found in many different parts of the circulatory apparatus. Whether seen in the serous membranes, muscular substances, or connective tissues, they give rise to some of the most noteworthy disturbances of the circulation. All these changes must be regarded as the reaction of the tissues to irritation, and the processes present a uniform type, varying without doubt in degree but not in kind. Such reactions are only possible in tissues containing blood vessels or in close connection with them.

The changes present two different aspects for observation, one of which may be termed exudative, the second proliferative. The exudative process is more particularly seen in connection with the blood vessels, and marks the earlier stages. The exudation of coagulable lymph takes place attended by the



presence of some leucocytes and red corpuscles. The proliferative process occurs at a later stage and is observed in the endothelium and connective tissue, as well as in the blood vessels. The connective tissue cells multiply, as do also the endothelial cells. The products of proliferation undergo a change into fresh connective or fibrous tissue.

These reaction processes differ considerably according as the seat of the process is upon a membrane, like the pericardium, which is rich in blood vessels; or one like the endocardium, covering the valves, which has few or no blood vessels; or in a muscular structure like the myocardium, in which the blood supply is extremely free. In the pericardium the dilatation of the arteries and the increased velocity of the blood, followed by dilatation of capillaries and veins with retardation of the current, marginal arrangement of leucocytes, and diapedesis, lead to exudation. In the endocardium covering the valves, such processes are greatly modified, not only by the small number of vessels ramifying in the parts, but by the mechanical arrangement of the cusps; in connection with the valves, nevertheless, considerable exudation may be seen, leading even to fusion of the cusps. The changes occurring in the myocardium are modified by the special structure of the tissue in which they take place. Acute processes usually produce enlargement of the nuclei, or even proliferation, but without karyokinesis. The muscle cells are swollen and lose their transverse striation; they are usually altered in outline and are probably softer than in health. There is a swelling of the intermuscular substance, which may be invaded by leucocytes and form the starting-point of an abscess, or may be the seat of newly-formed fusiformed cells going on to the production of fibrous tissue.

The further processes as regards the changes undergone by such a serous membrane as the pericardium may be noted. Part of the fluid exudate which is poured out coagulates, and may form a regular membrane upon the surface. The serous effusion which follows is due to the exudation of a fluid less coagulable, possibly on account of some inhibitory influence exercised upon it by products of bacterial activity. If there be great emigration or proliferation of leucocytes with a small tendency to coagulation, the exudate becomes purulent.



Many of the cells which are present in pus are dead or even degenerated, but numbers of them still preserve amœboid movements and act as phagocytes. In certain cases there may be excessive diapedesis of red corpuscles, possibly due in great part to the severity of the exciting cause or to lowering of the resistance of the tissues. It is to be remembered, however, that mechanical injuries or malignant invasion may be the cause of such hæmorrhagic exudations. The proliferative changes go on *pari passu* with some of those which have been considered, but no doubt they are best seen when the exudation is passing away. Such proliferative changes may be present to a smaller or a greater extent. They may be present only in slight degree, and recovery may take place by resolution. They may, on the other hand, be present to a much greater degree, when repair occurs by means of proliferation. In many of the stages of proliferative repair phagocytosis is still seen.

Occasionally these acute processes manifest destructive tendencies. The extent to which they take place depends upon the relative activity of the irritant and the reaction of the tissues. If proliferation is able to make headway, young connective tissue is formed and the process is brought to an end. If, on the other hand, the irritation is more powerful than the resistance of the tissues, ulceration results.

Some of the secondary results of such processes require also brief mention. The processes produced by organisms are apt to be followed by remote effects in distant regions. The organisms or some of their toxic products may not remain localised, but be carried from the seat of the lesion either by the lymphatics or the blood vessels. In croupous pneumonia, for example, the pneumococcus may be carried to the pericardium or endocardium. In endocarditis, streptococci may be carried to the kidney or other organ, and in this way secondary infection may be set up. Bacterial emboli may enter the venous channels at any point and find a temporary stopping-place on one of the cardiac valves, producing there infective endocarditis, whence a shower of bacterial emboli may be distributed through the whole body.

There are several chronic processes due to irritation which vary considerably according to the tissues in which they

take place, and to the extent of the irritation. In the case of a serous membrane, chronic irritation results in thickening, sometimes with a considerable amount of contraction, often with a large amount of newly-formed fibrous tissue, and occasionally with a deposit of lime salts.

Chronic processes taking place within the myocardium are characterised by the large amount of fibrosis which occurs. The fibrous tissue separates and compresses areas of muscle fibres, constituting what is commonly known as chronic interstitial myositis. Such chronic processes are essentially hyperplasias of the connective tissue.

The processes of repair in the higher animals are vastly inferior in their scope to those found amongst more lowly forms of life, inasmuch as regeneration only takes place by the production of certain elements of the tissue instead of the growth of entire organs. Epithelial and connective tissues are the results of reparative processes. In regard to the circulatory organs this is directly true, and destruction of any part is only made good by the growth of the less specialised tissues. The repair which takes place in the serous membranes may be largely homologous. In the muscular structures it can only be heterologous.

COMPENSATION.—Hypertrophy may be regarded within physiological limits as a process of adaptation; in pathological circumstances it must be looked upon as a process of compensation. All structures may increase in size under certain conditions, pathological or physiological, and if such enlargement takes place without any structural changes, the process is termed hypertrophy. Every part of the healthy body possesses considerable reserve of energy. In health, the demands made upon the various organs are far below their possible responses, and, if calls on their functional activity are sustained, they adapt themselves. Under abnormal conditions, the process of hypertrophy is produced by the power which the different structures of the body possess of responding to the demands made upon them by readjustment of the tissues, and so long as this process of hypertrophy is adequate all symptoms of disturbance may be absent. An excellent example of such readjustment is to be found in chronic cirrhosis of the kidneys attended by

increased thickness of the middle tunic of the arteries and hypertrophy of the left ventricle.

REPAIR.—Restitution or recovery may without doubt occur in serous membranes. Many cases presenting absolute proof of pericarditis are found at later dates to have no trace of any structural change. It is possible that the same thing may occur in endocarditis, and in another place attention has been given to the subject. When, however, such a structure as the myocardium undergoes structural alteration, absolute restitution is out of the question. In interstitial myocarditis, for instance, there is, as a result of the process, a development of fibrous tissue in which the newly formed tissue impairs the strength of the heart, and may produce the most serious symptoms.

### RESULTS.

Disturbances of the circulation are the results of a large number of individual factors, but when these are carefully analysed it is possible to arrange them in a limited number of groups. Disordered conditions of the circulation owe their origin to factors producing their chief morbid effects respectively upon the heart, the blood vessels, and the blood. It is no doubt true that many causes operate by influencing more than one, sometimes all, of these structural divisions of the circulatory mechanism, yet it is convenient, and even necessary, to classify the various agencies for the purpose of thorough investigation.

FROM DISTURBANCES CONNECTED WITH THE HEART.—The equilibrium of the circulation may be disturbed by many pathological factors having their origin in, or acting directly upon, the heart.

*Pericardial.*—Some of these causes interfere with the functions of the heart by pressure from without. The best example of this class is to be found in pericardial effusion. It is a well-known clinical fact that serous pericarditis causes considerable embarrassment to the course of the blood. Adamkiewicz and Jacobson have shown that in healthy conditions there is a negative pressure of 3·5 mm. Hg. in the pericardial sac. When there is any considerable effusion,

however, this negative pressure becomes positive; and Cohnheim proved by experiment how such a positive pressure interferes with the circulation. By connecting one of the veins of the neck with a soda manometer, and one of the carotid or femoral arteries with a mercurial manometer, Cohnheim provided the means of observing the effects of pericardial pressure on the venous and arterial flow. The pressure in the sac was varied by connecting the parietal pericardium with an oil manometer. The pressure in the sac might be raised to 30 or 40 mm. of oil without any change in the vascular pressures, but if the oil manometer showed a pressure of 60-70 mm. there was a fall of arterial pressure of about 20-30 mm. Hg. and a rise of venous pressure of about 60 mm. Na; an intrapericardial pressure of 100-120 mm. oil, produced a great fall of that of the artery, while that in the vein rose to a high level. By increasing the pressure of oil still further, the arterial curve fell to zero, while that of the vein rose higher still. If the experiment was not continued too long the normal conditions could be restored on relieving the pericardial pressure, the first effect being that the arterial curve rose to a higher level than previous to the experiment, before resuming its normal height.

Starling has repeated Cohnheim's experiment, with two modifications. In the first instance, he estimated simultaneously the pressures in the inferior vena cava, in the portal veins, and in the arterial system, under the influence of pericardial injections. In the second place, he observed the effects of such injections on the volume of the limbs. In the first set of experiments cannulae were connected with a splenic vein, an iliac vein, and a carotid or femoral artery. The cannulae attached to the veins were connected with manometers filled with a coloured solution of sulphate of magnesium, while the arterial cannula was connected with an ordinary mercurial manometer. After the attachment of the manometers, the chest was opened in the middle line, and a cannula tied into the pericardium, for the injection of oil by means of a graduated burette. In one such experiment the following results were obtained:—



	Carotid. Hg.	Splenic. MgSO <sub>4</sub> .	Iliac. MgSO <sub>4</sub> .
	mm.	mm.	mm.
Before injection of oil . . . . .	90	128	36
After 20 cc. oil . . . . .	90	128	36
" 40 " . . . . .	90	128	40
" 60 " . . . . .	90	128	58
" 70 " . . . . .	...	134	76
" 90 " . . . . .	56	160	124
" 100 " . . . . .	18	215	215
After escape of oil . . . . .	146	322	36
Later . . . . .	84	148	40

This experiment shows that but little influence was exerted on the pressures until 20 cc. of oil had entered the pericardium, and that the limits of compensation possessed by the heart and vascular system for the hindered diastolic expansion were only reached when 70 cc. of oil had been injected. The possibilities of diastolic expansion came to an end when 100 cc. of oil had been injected. Some of the results are rather difficult of explanation, and perhaps this is especially the case as regards the enormous rise in arterial pressure which occurred directly after the recommencement of cardiac activity. It is, however, probable that at the point when the heart stopped the arterioles were in a state of constriction, and that when the heart, richly supplied with blood from the distended venæ cavæ, propelled this into the arteries, it had to overcome the resistance in the arterioles.

The other series of experiments performed by Starling was devised in order to ascertain the effects of pressure changes on the volume of the limbs. An experiment similar to that above referred to was carried out, while at the same time the limb was enclosed in a plethysmograph. In this experiment it was found that, while the arterial pressure remained constant, practically no change occurred in the volume of the limb, although there was a rise of pressure both in the portal veins and vena cava. Whenever compensation became insufficient and the arterial pressure commenced to fall, the volume of the limb diminished. The volume of the limb, and probably the pressure in its smaller vessels, is therefore directly proportional to the arterial pressure, and is not altered by considerable changes in the venous pressures consequent on heart failure.



The conclusion seems to be obvious that even with vascular constriction the effect of heart failure must be a fall and not a rise of blood pressure in the capillaries and smaller veins of the limbs. This conclusion is at variance with the view usually accepted, for the most part on the authority of Cohnheim, that failure of compensation in heart disease produces a general rise of venous and capillary pressures in all parts of the body, at least in brief interferences with the conditions of the circulation. When they are long continued other influences come into operation.

Starling has further set himself to ascertain whether any of the conditions in heart failure might lead to plethora sufficient to raise the venous and capillary pressures in the limbs in the absence of any arterial rise, and has come to the conclusion that the results of a number of observations on cases of cardiac failure lend no support to the view that failing compensation is attended by plethora. The results tend rather to show that the amount of blood in the circulation is below the normal, and that there is a condition of hydræmia, not of plethora. The conception that the pressure in the capillaries and veins all over the body in failure of compensation is elevated must be renounced, as the pressure must follow the arterial pressure and be lowered.

There are other causes of interference with the heart functions by pressure from without. The existence of any considerable aneurysm or tumour within the chest produces a similar effect, and pleural effusion or pneumothorax has the same result. Such causes are operative by hindering the return of blood to the heart, and this is effected by interference with its aspiratory or suction-pump action. External causes may further interfere with the systole of the heart. In adherent pericardium, for example, there may be considerable hindrance to the contraction of the heart, in other words, the force-pump action is retarded.

*Endocardial.*—When the internal mechanism of the heart is disordered another group of causes comes into play. Valvular affections, by obstructing the outlet, or by allowing escape backwards, lead to much disturbance. In the case of any orifice which has been narrowed by disease, whether from endocarditis or

degeneration, there is a hindrance to the flow of the blood through it, and, unless the obstacle is in some way overcome, there must be a lessened current through the opening.

The valves closing the orifices of the heart may be incompetent from several causes. The cusps themselves may be in fault, on account of some endocardial change which has caused them to shrivel up. The orifices may, on the other hand, have become too large for the cusps to be adequate. It is possible, moreover, that when, without any stretching of the orifices, there is dilatation of the ventricles, the cusps cannot accurately close the auriculo-ventricular openings on account of lack of adaptation of the different parts of the mechanism—papillary muscles, chordæ tendineæ, and valvular segments. In both these lesions, obstructive and regurgitant, the outward flow of the blood is lessened—in other words, the force-pump action of the heart is hindered—by an obstacle to the onflowing current, or a loss of part of it backwards. The attractive force of diastole is also interfered with directly or indirectly; obstruction, at any rate at the venous valves, prevents the full aspiratory action, while regurgitation by the backward escape causes some of the blood to be drawn towards the heart more than once.

*Myocardial.*—There are also causes operative through weakness of the walls of the heart. Such processes are almost invariably secondary to diseased conditions elsewhere. To this class belongs myocarditis, both in its acute and chronic forms, but more commonly the latter; the various degenerations, fatty, pigmentary, and hyaline, as well as the cloudy swelling often present in acute diseases, and the fragmentation which may occur in them; together with simple debility of the muscle, from acute or chronic disorders of the nutritive functions. Disturbance of the coronary circulation is followed by inevitable changes in the muscular substance of the heart. If there is any diminution in the calibre of the coronary arteries, there is certain to be a degeneration of the myocardium, and if septic emboli reach their distribution, septic myocarditis is an invariable sequel. It has already been shown in the previous section that experimental pathology has so far thrown a somewhat uncertain light upon the facts of

sudden death and angina pectoris ; it is nevertheless an undeniable fact that the entire adequacy of the heart depends upon the integrity of the coronary circulation.

RESULTS OF THESE DISTURBANCES.—All processes which lessen the energy of the muscular substance of the heart have a twofold effect. They diminish the aspiratory or suction-pump action of the heart during diastole, and also, to a proportionate extent, lessen its expulsive or force-pump action during systole. It follows that such lesions as those just referred to, which give rise to a reduction of the energy of the heart, both as regards aspiration and expulsion, have effects of far-reaching consequence upon the entire circulation.

It has already been shown in the physiological section, that the energy produced by the heart appears partly as the potential energy of the distended arteries, partly as the kinetic energy of the moving blood. The work done by each cardiac systole may be estimated from the formula previously given—

$$W = BR \frac{Bv^2}{2g}$$

where  $W$  stands for work,  $B$  for amount of blood expelled at each systole,  $R$  for arterial pressure, and  $v$  for velocity of blood. As was previously demonstrated, velocity may be disregarded in considering pathological changes as affecting the work of the heart. Any real increase in its work can only be produced by an increase in one or both of the two factors  $B$ ,  $R$ , that is to say, in the amount of blood expelled, or the resistance offered, by arterial pressure. Most cases of heart disease present an increase in one or both of these factors. In aortic obstruction, for example,  $R$  may be largely increased. In aortic regurgitation, on the contrary,  $R$  may be diminished, but, as during diastole the ventricle receives blood from both sides, and in compensation the heart expels almost all the excess of blood which it has received, there is an increase in  $B$ . When obstruction is associated with regurgitation both  $B$  and  $R$  are increased.

The healthy heart has a large amount of reserve power, as Cohnheim has shown, and can therefore answer to greatly increased demands by doing more work. This power of

adaptation may be tested by the experimental imitation of pathological conditions. The resistance to be overcome by the heart may be increased three or four times without causing any change in the amount of blood expelled at each beat. A ligature may be placed round the pulmonary arteries or the aorta, and the lumen of the vessel may be reduced to one-third of its size without materially interfering with the arterial pressure. If a manometer, however, be connected with the cavity of the ventricle, it is found that the amount of energy which it is exerting is increased to three or four times its normal amount.

Instead of increasing the resistance to the outflow the inflow may be experimentally increased by destroying the semi-lunar cusps, in which case but little disturbance is produced in the blood pressure, although the amount of energy of the ventricle is greatly increased as tested by the manometer.

Experimental observations have shown that on exciting the peripheral end of the divided vagus, the heart is slowed, the diastole lengthened, and the amount of blood expelled at each systole augmented. There is nevertheless a fall of arterial pressure, and the increased outflow is not proportional to the diminished frequency of the heart; R and B being both lessened, the work done by the heart is therefore diminished.

On stimulating the nerves which reach the heart through the sympathetic system, the heart is quickened, the diastole shortened, and there is often a rise of blood pressure. In almost every case of such stimulation of the sympathetic system the outflow is raised above the normal, so that the work done by the heart is increased. These nerves may be thrown into action by the medullary centres by direct or reflex influences. The most important are probably those reflex influences taking their origin in the heart itself, which, acting reflexly on the medullary centres, bring about inhibition or acceleration of the heart, or a rise or fall of blood pressure. Starling points out that these reflex mechanisms are not so much directed to compensation of vascular disorders by increased efforts, as to sparing the heart by the production of some reflex effect counteracting the original disturbance. A sudden rise in arterial blood pressure, for instance, greatly increases the work of the ventricles,



but any such rise of arterial pressure is accompanied by a slowing of the heart in consequence of stimulation of the vagus centre. The stimulation may be partly the effect of the high pressure of the blood circulating in the brain, partly a reflex influence from the heart itself.

The power of adaptation possessed by the cardiac muscle is apparently closely associated with susceptibility to tension. Whenever increased work is thrown on the heart muscle the contraction is preceded or accompanied by increased tension of the muscle. In aortic stenosis, for example, the increased tension occurs during contraction. In aortic regurgitation the increase of tension is present just before the contraction in consequence of the increased diastolic filling of the heart. When other forms of contractile tissue are examined an increased tension or increased resistance acts as an additional stimulation, so that the contraction occurring under such conditions is more powerful and more extensive than under ordinary circumstances. The effect of this tension in increasing the energy of muscular contraction is more strikingly shown if the increase is applied before the beginning of the contraction. This subject has recently occupied the attention of Horvath, and his views will be fully discussed in the chapter dealing with hypertrophy. One most interesting proof of this is found experimentally. If the lower third of the ventricle of the frog be isolated physiologically by crushing a ring of tissue between it and the upper part of the ventricle, it will not beat again under normal conditions, but it may be caused to beat rhythmically on clamping the aorta, so that there is increased intraventricular pressure and augmented tension of the muscular fibres.

Dilatation of the cardiac cavity is a very common sequel to valvular disease. It may occur under conditions perfectly physiological. The experiments of Roy and Adami, as well as of Johansson and Tigerstedt have shown that the ventricles are never under any circumstances emptied by their contraction, and his researches have shown that the amount of blood remaining in the ventricle depends on, first, the resistance in the arteries, second, the diastolic filling of the ventricles. There is a constant increase in the systolic volume of the heart if



there be any rise of arterial pressure or any increase of the diastolic inflow.

In all these cases, whether there be diminished aspiratory or propulsive energy, the result is a diminution in the amount of blood in the arterial, and an increase in the venous system. It does not follow as a necessary consequence that because there is less blood in the arteries the pressure of blood within them will be diminished, nor is it necessary that the venous pressure will be raised because the veins contain more blood—the blood pressure depends upon too many factors for such effects to be the inevitable consequence of changes in the amount of blood. It is quite possible, as Thoma says, on the authority of Worm Müller, that the blood pressure may not be altered, notwithstanding a change in the amount of blood, since the vascular tone may be so modified as to equalise the alteration in the quantity of blood. As a matter of fact, however, the comparative distension of the veins and the relative emptiness of the arteries produces a considerable alteration in blood pressure, the arterial falling while the venous rises. The consequences of the venous distension is to allow the hæmostatic factors to overcome the hæmodynamic, and in consequence of the venous engorgement several effects become manifest. On account of transudation there is œdema into the dependent parts of the body and dropsy into the great serous sacs. From the slowness of the current the blood absorbs too much carbonic acid, and is deprived of more of its oxygen than is the case under healthy circumstances; as a consequence, cyanosis results. And also on account of the slowness of the circulation there is a longer period of radiation of heat from the surfaces; the superficial parts of the body, therefore, become cold. If the processes are unchecked, hypostasis is the inevitable result.

When subjected to long-continued engorgement the liver becomes enlarged and assumes the appearance commonly termed the “nutmeg liver.” In consequence of the venous stasis in the radicles of the hepatic vein, there is interference with the distribution of the blood brought by the portal circulation, in addition to stagnation of bile in the ducts. From the interference with their functions, simple atrophy and

fatty degeneration take place in the hepatic cells. From the fatty degeneration in the circumference of the lobule, it has a white border contrasting with the ruddy tint of the engorged hepatic veins and the yellowish hue of the obstructed ducts; hence the name by which this condition is commonly known. It has been held that in addition to these changes hypertrophy of the connective tissue arises, but, when this occurs, it is usually the result of concomitant causes, and it is probably, as Coats says, a mistaken view that cirrhosis of the liver arises simply out of passive hyperæmia.

Infarction of the kidney is frequently found. Infarcts are usually of a pale colour and dense consistence. From coagulation necrosis there is seldom much hæmorrhage, but a little commonly occurs at the margin, or even, if its area be small, the hæmorrhage may extend throughout the whole infarct, while, around it, there is a zone of hyperæmia.

When hæmoptysis occurs on a large scale, it is almost invariably the result of rupture of the capillaries in the alveoli. A great amount of discussion has been devoted to hæmorrhagic infarction of the lung, into which it is impossible in this connection to enter. It may, however, be said that such infarction appears from the researches of Panum and Cohnheim not to be of embolic origin, but usually from rupture of the alveolar capillaries, the so-called pulmonary apoplexy, as it is often absurdly termed. The opinion that pulmonary infarct takes its origin in emboli arising from coagulation within the right cavities of the heart is founded on simple speculation, and is, according to Hamilton, a theory of the most unwarranted character. My own opinion is, however, that it may often be produced by thrombosis. In the majority of cases the vessel is found to be blocked by a coagulum.

Effusion into the serous sacs takes place under conditions somewhat similar to those which obtain in the subcutaneous tissues, but there are necessarily some points of difference. Starling and Leathes attempted to produce pleural effusion by an increase in the capillary pressure. As the blood returning from the pleura has several different channels, the only possibility of producing a rise of capillary pressure is by the injection of large quantities of normal saline solution into

the circulation, but, in addition to employing this method, they also made at the same time an attempt to increase the rise of pressure by obstruction of the vena azygos and thoracic duct. The result was a large amount of œdema in the retro-peritoneal tissue and in the posterior mediastinum, but the pleural cavities only contained 2 or 3 cc. of fluid. The injection of small quantities of jequirity into the pleural cavity gave rise to endothelial changes, and the injection afterwards of large quantities of normal saline solution into the circulation produced a great transudation of fluid through the injured capillaries into the pleural cavities, and death by asphyxia. The hydrothorax of heart disease seems, therefore, like the œdema of the limbs, to be dependent in the first instance on an increased permeability of the vessel, produced by the stagnation and poor quality of the blood. The peritoneum has two separate capillary systems, one or both of which may produce ascites:—the capillaries of the spleen and alimentary tract forming the radicles of the portal vein, and the hepatic capillaries ending in the hepatic vein. From the experiments already described it has been shown that there is a considerable difference as regards their permeability, as well as in the effect of various changes of the circulation on the pressure within them. Cardiac failure, for example, produces a rise of pressure in the hepatic capillaries, and a fall in the intestinal capillaries. As regards changes in the portal system of capillaries, it seems probable that the result of increased flow of lymph depends on the condition of the endothelium of the peritoneum. Ligature, for example, of the portal vein causes a great increase of the flow from the thoracic duct, but very slight transudation into the peritoneal cavity. If, however, the permeability of the capillaries is increased, as may be done, by plunging some coils of intestine into hot water, or even distilled water at the temperature of the body, there is a considerable transudation into the peritoneal cavity, although the lymph flow from the thoracic duct is scarcely affected. It seems probable that in heart disease the most important source of ascites is the liver. In one of Starling's experiments ligature of the thoracic duct and obstruction of the inferior vena cava above the liver, followed by the produc-

tion of hydræmic plethora through the injection of 15,000 cc. of normal saline solution, produced 100 cc. of ascites. In another experiment the hepatic lymphatics were ligatured as they emerged from the portal fissure and a hydræmic plethora produced in the same way; this was followed by the presence of 230 cc. of ascites. We know that the capillary pressure in the liver is considerably increased in heart disease, and we are warranted in concluding that the chief source of ascitic fluid in such affections is to be sought in the capillaries of the liver.

COMPENSATION FOR THESE DISTURBANCES.—The heart, having considerable reserve of energy, in most cases is able to meet minor degrees of disturbance of its functions successfully. If such disturbances, however, are permanent, so that the organ is subjected to continuous stress, certain changes in function and structure take place, whereby it is enabled to overcome the strain. The processes by which these changes are brought about unfold themselves gradually, and they are, therefore, not always obvious. Many of them may, nevertheless, be traced during their gradual evolution, and there are but few gaps in our knowledge which require to be filled up by inference instead of by observation. The various changes which ensue are summed up under the term "compensatory."

It has been said above that by means of its reserve of energy the heart is able to deal successfully with lesser disturbances of the circulation, and Cohnheim and his followers proved that even considerable changes in the mechanism of the heart may be produced without disturbing the equilibrium of the circulation. It is a subject of the highest import that after destruction of the aortic cusps in the dog there was no change in the arterial or venous pressure. On the production of stenosis, whether of the aortic or pulmonary orifice, or, to be more correct, of one of the great arteries immediately beyond these cusps, the arterial and venous pressures were found to remain completely unchanged until the stenosis was carried to a very considerable extent. But when this point was passed the arterial pressure underwent a sudden fall, while the venous pressure rapidly rose. From such experiments, which have been repeated more recently by



Rosenbach, much has been learned. They prove absolutely that the heart is endowed with a considerable margin of energy. They show, moreover, the influence of destruction on the character of the individual pulsations. The narrower the lumen of the vessel, and the higher the cardiac pressure, the more amplitude do the single contractions acquire. At first, as Cohnheim showed, these greater pulsations do not occupy more time than the normal pulsations did before the commencement of the experiment, but, when they exceed certain limits, they cannot be maintained without more time being occupied by each of them; *i.e.*, the pulsations become larger, and also, at the same time, less frequent.

But the consideration of the artificial stenosis places before us the explanation of some other points connected with compensation. The extent of the stenosis may be increased, and the resistance, consequently, intensified, yet the heart overcomes those and expels as much blood as previously, so that the mean arterial and venous pressures are maintained at their normal height. But when the resistance becomes too great, so that the contractions of the heart are unable to overcome it completely, the circulation comes absolutely to an end.

The experiment upon positive pericardial pressure described by Cohnheim and previously referred to, stands in marked contrast to those artificial valvular lesions. As previously said, it shows that when there is a hindrance to the afflux of blood to the heart, there is from the first a change in the relative arterial and venous pressures. The explanation of this is simple. When a smaller amount of blood is allowed to reach the heart and to be sent on into the arterial system, there is of necessity a state of relative overfilling of the venous, and of relative underfilling of the arterial vessels. And there is, in this case, no adequate provision to meet its requirements. It is abundantly clear, from a consideration of the relative force of suction and of expulsion, that a very much smaller disturbance will interfere with the former than with the latter.

In the case of lesions of the orifices and valves, there are some other considerations which require attention. In such lesions provision is made for a larger quantity of blood in the



chambers of the heart. The blood is expelled from the heart less frequently, although with greater force, and it therefore must follow that a larger quantity is allowed to accumulate in the chambers during diastole. The heart must, therefore, undergo a considerable amount of distension, just as happens in the case of all hollow viscera, such as the stomach, when their contents are retained for a longer period than usual.

In order to overcome the various obstacles with which we have been dealing, the heart acts with more energy. In all cases where the heart is able to survive any damage it may have sustained, it must have been primarily endowed with that reserve of energy which has already been spoken of; and if the disturbance to the circulation were transient, this in itself would be sufficient to maintain the balance. Most of the lesions to which the heart is subject are permanent in their effects, and compensation is carried out by means of another process. This is hypertrophy of the walls of the heart. Speaking generally, it may be said that hypertrophy may be total or partial, according as the disturbance affects the whole heart or only a part of it. When there is a call for additional energy on the part of the entire heart, as in synechia of the pericardium, the walls of each chamber become increased in thickness, in consequence of the increased energy which it manifests. If there be, on the other hand, a disturbance affecting only one chamber, that particular portion chiefly undergoes hypertrophy. It cannot be said that each individual chamber is alone affected, for, as Gairdner showed long ago, such a thing as hypertrophy affecting one chamber alone is practically unknown. When there is hypertrophy of one side of the heart, the other is usually to some extent involved. It is unnecessary in this place to tread the well-worn path of the usual series of events in compensation for valvular disturbance. The entire subject will be discussed in a future chapter.

When there is any lesion at the aortic orifice, whether it be obstructive or incompetent, in its nature, the additional stress so produced falls of necessity for the most part upon the left ventricle, and it therefore becomes hypertrophied to meet the obstacle. If there be a lesion of the mitral orifice or its cusps, the walls of the left auricle become thicker. In the

case of the pulmonary orifice or valves, it is the right ventricle which increases in bulk; and when the tricuspid orifice or its valves are abnormal, the right auricle undergoes hypertrophy. When there are combined lesions of the orifices or valves, there are of necessity combinations of hypertrophy.

Hypertrophy is only possible when the nutrition of the heart is good. It can only, therefore, occur when there is an adequate supply of healthy blood, and this necessarily means that the blood itself must be healthy, and that the coronary arteries must be sufficiently pervious to allow an adequate supply of such healthy blood to reach the heart muscle. The process of hypertrophy is therefore subject to individual limits, when nutrition is at a low level it may fail to appear; when there is any inherent narrowness of the lumen of the coronary arteries its extent is circumscribed. In every instance, the end at last comes when the heart has outgrown the nutritive possibilities granted by the conditions present. It has often been held that there is no hypertrophy without a certain amount of fatty degeneration. This, as Hamilton has pointed out, is probably a grotesque exaggeration; yet it cannot be gainsaid that, at the end of a long existence of hypertrophy, some fatty degeneration steps in. It is the only argument in favour of the view recently expressed by Meigs that compensatory hypertrophy has no existence. When a heart has outgrown the nutritive possibilities with which it is endowed, there is said to be failure of compensation. This may be general or it may be local.

FROM DISTURBANCES CONNECTED WITH THE BLOOD VESSELS.  
—It has already been seen that the blood vessels are endowed in various degrees with contractility and elasticity. The contractility of the blood vessels entirely depends upon the state of the muscular coat. As was seen in a previous section, the muscular tissue of the blood vessels is characterised by a high degree of tonicity, and a considerable development of rhythmic action, while rapidity of contraction is almost absent. The elasticity of the blood vessels is not to be regarded as an independent force, but simply as a means of storing energy in a potential condition which may be liberated in the kinetic form when required.

It has further been seen that the blood vessels are supported in various degrees by the tissues in which they run, according to their different situations. This fact, long ago pointed out by Donders, and recently insisted on by Hamilton, is apt to be lost sight of in considering the functions of the blood vessels. The different degree of support afforded by the tissues in different parts of the body explains how it is that certain blood vessels are more liable to destructive changes than others. In the brain and lungs, for example, the vessels have not, by any means, the same amount of support as they have in the more solid or firm viscera, and it is in these situations that vascular disasters are most common.

As part of the natural processes resulting from advance in years, the contractility and elasticity are apt to diminish, while, at the same time, the support afforded by the tissues fails on account of shrinkage. It is perfectly true that such events take place at very different periods in different persons: in one the blood vessels appear to be endowed with such a degree of vitality as to exist in a comparatively healthy condition until an advanced age has been attained; while in another the blood vessels undergo retrograde changes in comparatively early years. This well-known fact has given rise to the epigram that "a man is as old as his blood vessels."

The facts which we know as to changes in the condition of contractility and elasticity are mostly connected with conditions in which these are diminished. Long continued stress of the blood vessels induces thickening of the intima, wasting of the muscular coat, and stretching of the adventitia. In consequence of these changes both contractility and elasticity are gradually lost. This has been well recognised since the days of Boerhaave, who described changes in the walls of the great blood vessels in deer which had been allowed to live in a wild condition, while the same animals reared in confinement showed no such changes. These facts, accepted and expanded by numerous writers, form the earliest observations upon this subject.

When the normal properties of the arteries are interfered with, there is a natural tendency by which some of the effects

are minimised; an increased growth of fibrous tissue takes place, constituting the process known as fibrosis. It must be regarded as a compensatory change, whereby the walls of the blood vessels are strengthened in order to support the stress which they have to endure. Thoma holds that the result of these changes is an increase of elasticity. This has been objected to by me. What occurs is that greater rigidity takes the place of lessened elasticity. It is in truth a singular conception that increase of elasticity should be the precursor of dilatation or rupture of the arterial walls.

In several diseases there are characteristic changes in the blood vessels which are scarcely to be regarded as belonging to the compensatory category. In the uric acid diathesis there is undeniably a great tendency to increase of fibrous tissue, and later to the deposition of lime salts in the walls of the blood vessels. In the tertiary stage of syphilis, and in some chronic renal conditions, there is also a characteristic endarteritis obliterans. Both these processes involve, at a later stage, various interferences with the circulation.

Elasticity and contractility cannot be held to attain any point of development at any time above the normal, but, as was seen before, both are at their highest level when the blood vessels are full and high pressure is present.

We know much less of conditions in which peripheral resistance is diminished than of those in which it is increased. We are able to observe, nevertheless, that, just as external warmth dilates the blood vessels and lowers the blood pressure, so when the initial stage of pyrexia has passed away the resistance is diminished. Loss of blood and wasting diseases also lead to a similar reduction of resistance.

In considering the changes in the blood vessels, we have hitherto dealt only with conditions having their origin in these vessels. It is necessary to cast a rapid glance at some of the changes in which, from alterations in the structure of other organs, modifications of the circulation are produced through interference with the blood vessels. Since the writings of Bright it has been recognised that changes in the circulation are produced by chronic disease of the kidney, and, as was shown by Gairdner, there is considerable inter-



ference with the circulation in chronic bronchitis and emphysema. Other pulmonary conditions, such as induration, silicosis, and collapse, also interfere with the circulation in the blood vessels. It is a most interesting fact that in phthisis pulmonalis there is comparatively little interference, and this has received adequate explanation by the experiments of Lichtheim. Lichtheim found that by tying the left branch of the pulmonary artery there was absolutely no change in regard to the pressure of the blood in that vessel. He was able even to occlude several branches of the right pulmonary artery before such a change ensued. We may, therefore, assume that nearly three-fourths of the sectional area of the pulmonary blood vessels may be absolutely obliterated without producing any change in the blood pressure; the reason for this is not far to seek—it depends upon the remarkable ease with which the pulmonary blood vessels dilate. In Lichtheim's experiments, the whole of the blood normally passing through the entire pulmonary vessels passed with perfect ease through the third or fourth part of these blood vessels not interfered with by his experiments.

The effect of the various changes which have been considered is to throw an extra amount of work upon the heart, in order to overcome the increased resistance, and the effect upon the heart is hypertrophy. In their simplest form, *i.e.* loss of arterial distensibility, elasticity, and contractility in elderly people it explains why the heart, alone of all the viscera in the body, goes on progressively increasing in size with the advance of years, as was first shown by Bizot and afterwards explained by Perls.

Some of the special effects which have just been referred to have been carefully studied by Hamilton, and will be fully discussed elsewhere.

When the peripheral resistance is diminished, the heart, as is well known, has a tendency to race, and unless it be checked it is liable to run down. The nervous system in such cases, however, fortunately steps in and relieves the heart from the excessive waste of energy involved in continued increase of rate. And when this is not the case, if the inhibitory nerves do not reduce the rate of the heart within moderate limits,

that organ rapidly undergoes destructive changes. It is well known that in such febrile diseases as enteric fever, a high pulse rate is a prognostic sign of evil omen.

When arterial sclerosis is found throughout the body the coronary arteries of the heart frequently undergo the same change, and if it is sufficiently extensive to produce diminution of the blood supply to the heart, degenerative changes result. The most common of these degenerative changes is fibrosis. But fatty changes are also of frequent occurrence, either alone or in combination with a certain amount of fibroid degeneration. The results of experiments on the coronary arteries do not afford us much assistance in studying these chronic changes, for they are not only, as was already seen, somewhat difficult of explanation in themselves, but they also cannot be held to throw any light upon a gradual and long continued process such as coronary sclerosis.

One other effect of vascular changes must be referred to. The distribution of arteries to the papillary muscles, as originally shown by Swan and recently insisted on by Fenwick and Overend, is terminal in arrangement, and, if there be any change in one part of the arterial supply, there is no possibility of compensatory supply from adjacent vessels. This must be regarded as the reason for the frequency with which the papillary muscles undergo degenerative changes in many diverse conditions.

FROM DISTURBANCES CONNECTED WITH THE BLOOD.—The condition of the blood constantly varies in consequence of the tissue changes throughout the entire system. It is undeniable that a condition of oligæmia may be present in consequence of direct loss or continuous drain. It must be admitted that an opposite condition, polyæmia, may be produced in conditions involving an excess of production over loss. This latter condition of polyæmia or plethora is, however, rather suggested than determined, since much more than the total quantity of blood at any time present in the body could be easily accommodated in the veins. It seems absolutely certain that conditions of oligæmia and polyæmia are only transient, the various processes connected with the circulation being amply sufficient to restore the normal balance with great rapidity.

Changes in the number of blood corpuscles and of the hæmoglobin contained by the red corpuscles are of very much more importance than alterations in the total amount of the blood. We may speak of oligocythæmia in the sense of a diminution of the blood corpuscles without distinguishing between the relative diminution of either form, and of the converse condition, polycythæmia, when there is an increase in the number of the corpuscles. The former condition is produced as a symptom of many wasting diseases, the latter condition is usually the result of backward pressure.

The hæmocytes or red blood corpuscles not merely vary within wide limits in regard to number, but they also differ considerably in size and form. Megalocytes or giant corpuscles, microcytes or dwarf corpuscles, as well as poikilocytes or irregularly formed corpuscles, are often present in different varieties of oligocythæmia. The leucocytes or white blood corpuscles have a wider range of variation, both in number and size, than is found in the case of the red corpuscles.

The hæmatoblasts or blood plates are reduced or increased in number in a great many different conditions, but their relations are still in doubt.

The amount of hæmoglobin contained in the red blood corpuscles is diminished in a very large number of diseases, while it can scarcely be said to be increased except in the convalescence which follows acute diseases, and as the result of backward pressure from primary or secondary changes in the heart.

It has already been shown experimentally that the heart beat is modified by the quantity and quality of blood passing through it. If the heart, for instance, be washed out with any indifferent fluid its pulsations fall into groups and it finally comes to stillstand, thus throwing light upon the occurrence of intermission and asystole. If, after all the pulsations have been arrested, the heart is again fed with blood, it begins once more to beat normally. It has also been shown experimentally that, if the blood be acidulated, there is an increased expansion of the cardiac muscle, while if it be rendered more alkaline the expansion is lessened. The effect upon the heart of conditions in which the blood contains less nutriment is to

impair its energy. The cavities tend to dilate, the walls become atonic—in short, dilatation is inevitable.

Upon the blood vessels the effect of impoverished blood is to impair their nutrition, and, therefore, to lessen their contractility and elasticity.

But, in addition to such effects upon the heart and upon the blood vessels, a diminution of the nutritive properties of the blood is also of importance. There can be no doubt that, when the blood does not contain the elements necessary for active tissue change, the *vis à fronte* must be reduced. This is, probably, the explanation of the increased blood pressure often observed in conditions where the blood has been impoverished.



## CHAPTER IV.

### SEMEIOLOGICAL.

THE appearances presented by patients suffering from cardiac disease vary so widely that it is no easy task to arrive at any generalisation with regard to the subject. Between the pale tint of aortic incompetence and the dusky flush of mitral obstruction there is a wide gulf; there also exists an immense difference between the translucent pallor of profound œdema and the deep lividity of intense cyanosis. Inasmuch as a good many factors are concerned in the production of such changes in the complexion, it nevertheless often happens that two lesions, practically alike in nature and extent, may give rise to singularly diverse appearances.

The expression is often apathetic when circulatory disturbance has reduced the activity of the cerebral cells; it is frequently anxious when breathlessness and palpitation are prominent symptoms; it is always apprehensive in severe cardiac pain.

The attitude is occasionally of some use from the diagnostic point of view. In severe dyspnœa it is impossible for the patient to assume the recumbent posture. Cardiac pain causes a tendency to assume a position as rigid as possible. In thoracic or abdominal aneurysm the patient sometimes strives by bending forward to relax the parietes as far as possible, and in this way to relieve the symptoms of pressure.

It is well known that the symptoms of which patients complain, even in well-marked cases of circulatory disturbance, are often absolutely unconnected with the heart, and the more special cardiac symptoms require to be sought for. The

relation existing between different groups of symptoms fluctuates extremely, and it is not easy to make any general statement in regard to the degree of frequency with which these make themselves manifest. There can be little doubt, however, that the most frequent complaints expressed by patients suffering from cardiac disease are connected with the respiratory processes—breathlessness, especially on exertion, as well as cough and expectoration, being extremely common symptoms. Symptoms connected with the subcutaneous textures and the urinary functions are probably next in point of frequency, although various digestive symptoms are almost equally common, such as want of appetite, nausea, and vomiting, along with alterations in the intestinal functions. The nervous system frequently shows disturbances like giddiness or faintness, while overpowering drowsiness or troublesome insomnia are also common. Any or all of these symptoms may be associated with others more directly connected with the heart, such as pain, or palpitation, and fluttering or trembling sensations.

It is impossible in a systematic work to do otherwise than consider the symptoms of disease in groups. They will therefore be examined and analysed according to the systems by which they are manifested. It must, however, be clearly kept in view, not only that many symptoms have both subjective and objective aspects, but also that those mainly belonging to one system may be so closely interwoven with another group as to be separated therefrom only with extreme difficulty. The line of demarcation is therefore somewhat uncertain, and the discussion of a symptom belonging to one class will often be found to necessitate the consideration of matters connected with other groups. In the following pages the symptoms directly connected with the circulation will be considered in the first place, and thereafter the effects of circulatory disturbances will be traced throughout the other systems of the body. So far as is possible these symptoms will be not only described but also explained. It therefore follows that this section must inevitably overlap to some extent those devoted to physiological and pathological considerations.

## SYMPTOMS CONNECTED WITH THE HEART.

The pulsation of the heart was observed by most of the ancient writers on medicine, and Herophilus discovered the fact that its movements were simultaneous with those of the pulse. The descriptions of these old authors are, however, so intermingled with the fanciful ideas of the times as to be of comparatively little use, and it is not until we reach the observations of Harvey, Lower, Lancisi, and Senac that results based upon the inspection of the chest come to have any real value. The gradual acquisition of our knowledge in regard to the appearances presented by the præcordia as observed by the eye, will be noted as the various subjects are discussed in the following pages.

**INSPECTION.**—The præcordia or portions of the thoracic wall lying in front of the heart have already been described from the anatomical standpoint. The form varies considerably, not merely in the two sexes, but in consequence of age and individual peculiarities, but it may be said that as a general rule the præcordia are characterised by some flattening over the region of the sternum with a slight elevation on either side of it, which is rather larger on the left than on the right. By inspection information is gained as regards the form of the chest and the movements which it undergoes.

*Alterations in Form.*—Changes in the form of the chest are not produced by cardiac affections to any very considerable extent. It occasionally happens, however, that hypertrophy of the heart produces a certain amount of bulging of the præcordia, and this is more particularly the case if the hypertrophy occurs during early years. Thus it comes about that in congenital heart disease a distinct forward projection may be observed. In pericarditis attended by considerable effusion there may be bulging of the intercostal spaces—this, nevertheless, is one of the rarer manifestations of the disease. Projection of the thoracic parietes may be produced by aneurysm. Aneurysm of the ascending portion of the arch of the aorta gives rise in many instances to an obvious tumour in the region of the second and third right intercostal spaces, and it not infrequently happens that the adjacent costal cartilages or

ribs form part of the mass. Aneurysm of the transverse portion of the arch of the aorta produces a general projection of the upper part of the sternum and adjacent costal cartilages and intercostal spaces, which is not by any means easily distinguished from some of the alterations in form produced by pulmonary disease, unless distinct pulsation should be present in the region.

*Cardiac Movements.*—The cardiac impulse may be observed by inspection, but the facts so ascertained require to be verified, amplified, and corrected by the employment of palpation. In perfect health the only impulse perceptible over the præcordia is that commonly termed the apex beat, and this even is in many normal conditions by no means distinct. It is produced by the impact of some part near the anatomical apex of the heart upon the chest walls. The part of the heart which thus comes in contact with the parietes may be either the left or the right ventricle, according to circumstances. When the right ventricle is dilated and hypertrophied, it is obvious that it must displace the left ventricle backwards, and the position of the apex beat in such cases is quite in accordance with this fact.

The production of the apex beat has been a fertile source of discussion. By Alderson, Gutbrodt and Skoda, Jahn, and Hiffelsheim it has been regarded as produced by the recoil of the heart in consequence of the jet of blood sent into the aorta; by Kiwisch and Ludwig it has been considered as entirely caused by the change of shape of the heart itself; by Kornitzer it has been attributed to the somewhat spiral arrangement of the great blood vessels at the base of the heart; while by Aufrecht it has been held to have its origin in a flattening of the aorta during the outpouring of the blood. It is probable that each and all of these views contain some element of truth; but beyond question the greatest factor in the præcordial pulsation is the alteration of form which has already been discussed in the chapter devoted to physiological considerations.

The position of the apex beat, in the youthful adult, is in the fifth left intercostal space, between  $2\frac{1}{2}$  and 3 inches from mid-sternum. In the child it is very common to find the apex beat occupying a higher position, even in the fourth intercostal space relatively farther also from the middle line, while in the



aged it frequently occupies the sixth intercostal space, somewhat nearer the mid-sternum than in the normal standard. The apex beat undergoes some passive changes in position. With forced breathing it moves downwards and upwards respectively with inspiration and expiration. It further undergoes an alteration in position when the posture is modified. While lying on the right side the heart moves nearer to the mesial plane; and, on the other hand, while lying on the left side it passes farther away from that plane so as to approach the axillary line.

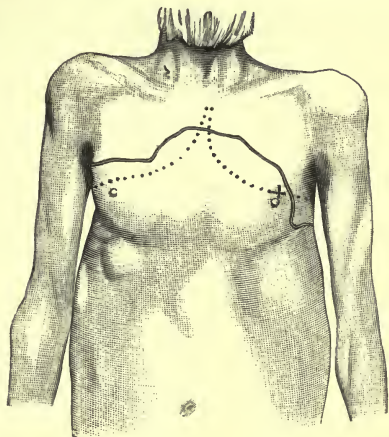


FIG. 17. — Upward displacement of thoracic organs in ascites. The dotted line gives the margins of the lungs; the double continuous line the upper borders of the heart and liver; the cross marks the apex beat.

In consequence of abnormal conditions the position of the apex beat may undergo considerable alteration in its position.

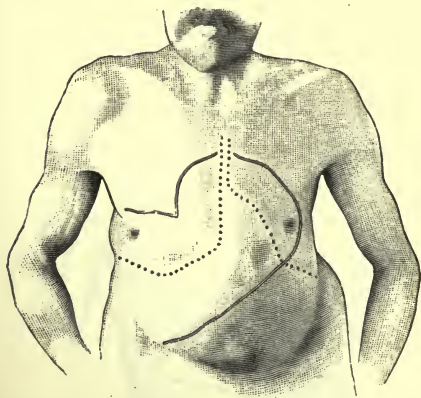


FIG. 18. — Upward displacement of the heart in scoliosis. The dotted line shows the margins of the lungs; the continuous line the cardiac and hepatic dulness.

In the presence of ascites, meteorism, tumours, or any condition whereby the contents of the abdominal cavity are increased to any considerable extent, the heart tends to be moved upwards. Such a condition may be seen in the illustration (Fig. 17), which represents the apex beat, along with the extent of cardiac dulness in an old-standing case of ascites.

A similar change of position may occur in

spinal curvature, as is shown in Fig. 18.

When the right pleural cavity is occupied by extensive

effusion, or much air, the apex beat is moved farther to

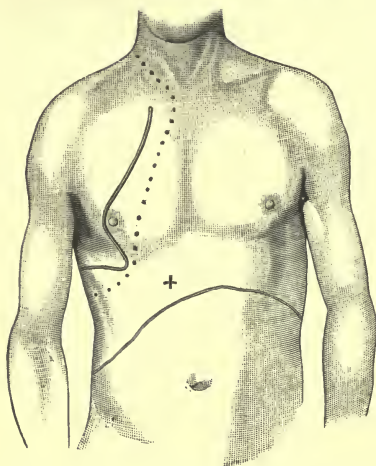


FIG. 19.—Displacement of the heart to the right in plenisy of the left side. The dotted line marks the anterior border of the right lung; the upper continuous double line the right border of the heart and the upper margin of the liver; the lower continuous double line the inferior limit of the dulness of the liver and the pleural effusion; the cross the apex beat.

the left than the normal; the same change in position is also produced if there be any considerable retraction of the left lung, such as is common in fibroid phthisis, and sometimes occurs as the result of interference with the root of the lung. In pneumothorax on the left side or extensive fluid effusion into the left pleural sac, the heart is often moved far over to the right side so as to reach at times the mammillary line.

This is well shown in illustrations 19 and 20.

A similar change of position occurs in consequence of retraction of

the right lung, as is shown in Fig. 21. The heart may be displaced downwards by various thoracic conditions, some of which are connected with the circulatory system, while others are produced by conditions of the thoracic contents other than circulatory organs. In cases of emphysema of both lungs the heart is displaced downwards; but, as will shortly be shown, the apex beat often tends to disappear entirely in consequence of the inter-

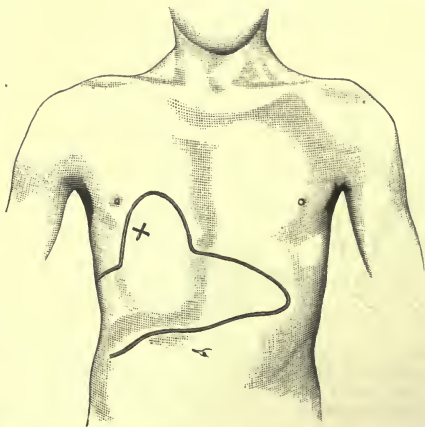


FIG. 20.—Displacement of the heart in left pneumothorax. The double line marks the position of the heart and liver; the cross the apex beat.

position of the distended lung between the heart and the parietes. In aneurysm of the arch of the aorta at almost any point the heart tends to be displaced downwards as well as outwards; it has, however, to be borne in mind that in such conditions lesions of the aortic cusps are extremely common, and their results upon the left ventricle have to be taken into account as in part responsible for the changes in position. The incidence of dilatation and hypertrophy gives rise to alterations in the position of the apex beat. In such affections of the left ventricle, the long axis of the heart is increased, and, as might be

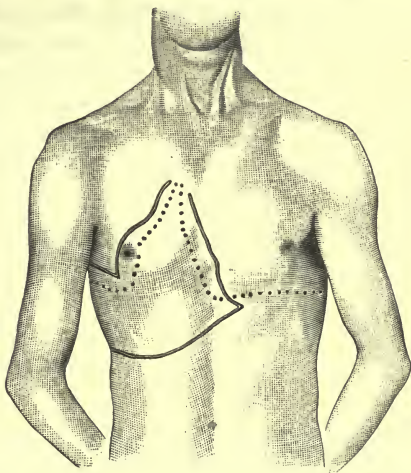


FIG. 21.—Displacement of the heart to the right from old fibroid changes in the right lung. The dotted line shows the margins of the lungs; the continuous double line the heart and liver dulness.

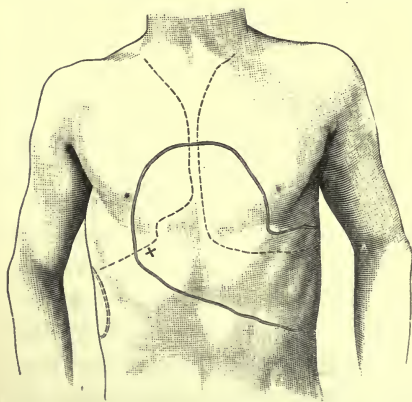


FIG. 22.—Complete transposition of the viscera. The double lines show the dulness of the heart, liver, and spleen. The dotted lines mark the margins of the lungs; the cross the apex beat.

expected from the examination of such hearts after death, the apex beat during life is found to be displaced, chiefly downwards, so as to reach even the sixth intercostal space, and only slightly outwards. In dilatation and hypertrophy of the right ventricle, the breadth of the heart is increased to a greater extent than its length, so that the chief alteration in the apex beat is one in an outward direction without much displacement

downwards. It must here be noted that in consequence of the

enlargement of the left ventricle the apex beat is very commonly produced by part of that ventricle, and it is therefore far from coinciding with the anatomical apex of the heart.

In addition to all these changes in the position of the apex beat there is one still more striking. In conditions of congenital transposition of the viscera the apex beat, instead of occupying the left half of the thorax, is found upon the opposite side, as may be seen in the accompanying illustration (Fig. 22). It represents the outline of the heart, lungs, liver, and spleen in a healthy schoolboy of sixteen, who was kindly sent to me by Dr. Cattanaeh.

The extent of the area over which pulsation can be seen is subject to alterations, more in consequence of conditions extrinsic to the heart, than connected with the heart itself. The area over which the apex beat can be seen is much larger in those who are thin than in those who are fat. It is also much increased when from any cause there is retraction of the left lung, while, on the other hand, it may be completely obliterated by the presence of such a condition as emphysema.

Pulsations of the præcordia may take their origin in movements of other parts than the apical region of the heart. One of the most common of these is the impulse often seen in the second left intercostal space. Movements in this region have been the cause of considerable discussion, as will be described in the sequel; but it may safely be asserted in this place that they are produced by the movement of the conus arteriosus or pulmonary artery during the systole of the right ventricle. A pulsation is occasionally, but very rarely, to be seen to the right of the sternum in the third and fourth intercostal spaces in consequence of movements of the right auricle. Much more common is a pulsation in the epigastrium, which may take its origin in several distinct causes. It may be caused by the throb of the right ventricle, communicated through the diaphragm in cases of dilatation or hypertrophy of that portion of the heart. It may be produced by an expansile pulsation of the liver in consequence of a reflux from the right chambers of the heart in cases of tricuspid incompetence, or it may be caused by the aorta, either by a simple exaggeration of its pulsation, or in consequence of an aneurysm.



Thoracic pulsations in the vicinity of the præcordia may also be produced by aneurysm of the aorta. Such pulsations occur in the situations which have been above referred to as the site of elevations of the surface.

The rhythm of the apex beat may be roughly estimated by the eye, but this is one of the points in regard to which the results of inspection require to be corrected by palpation. It is nevertheless possible to judge approximately as to the regularity or irregularity of the cardiac pulsations, and it may even be possible to determine whether the several heart-beats give rise to an equal amount of displacement.

One point yet remains to be noted. A retraction of the interspaces around the apex beat may be observed in certain conditions. Chief amongst these is obliteration of the peri-



FIG. 23.—Cardiogram from a case of adherent pericardium. The details of the case are to be found on p. 363.

cardial sac by adhesions, but in the total absence of such conditions there may be an indrawing of the interspaces, as, for instance, in examples of considerable cardiac hypertrophy. Skoda and his followers, *e.g.* Guttmann, describe a retraction of the apex beat in pericardial adhesion. This, however, is an absurdity. We know that the long axis of the heart is always lengthened during systole, and an indrawing of the apex beat is out of the question. The tracing (Fig. 23), taken from the apex beat in a well-marked case of pericardial adhesion, accompanied by great retraction of the interspaces surrounding the apex, shows quite distinctly that the apex beat is as usual a forward thrust and not a retraction.

**PALPATION.**—By means of palpation the facts observed by inspection may be verified and corrected, while additional phenomena may at the same time be determined.

*Form of Præcordia.*—The form of the præcordia may be

more fully investigated by adding the results of palpation to those obtained by inspection, and palpation may also, in cases where there is bulging of the intercostal spaces, give some indication as to the medium that has produced the bulging.

*Position of Cardiac Impulse.*—As regards the position of the impulses and their exact time-relations, palpation is of the greatest service, inasmuch as it not merely brings into greater prominence those which have been seen on inspection, but, furthermore, reveals the existence of degrees of pulsation too small to be detected by the eye. On palpating the cardiac region in a healthy person, a diffuse shock is felt, which, however, culminates in a sustained throb in the region of the apex. In those who are not very thin this is all that can be felt, but, when the thoracic parietes are slender, the sustained throb may be followed by a distinct, sharp jerk, produced by the recoil of the blood upon the sigmoid cusps during diastole. In a large proportion of individuals this distinct jerk can only be felt at the base of the heart, and more particularly in the second left intercostal space; in others, however, it may be felt over a wide area, including the apical region.

*Character of Impulse.*—In many instances a distinct movement somewhat like the peristaltic wave may be felt passing over the præcordia. It occurs in thin persons with vigorous hearts, but it is more characteristic of those cases in which from retraction of the left lung the heart is very largely uncovered.

*Force of Impulse.*—Alterations in the force of the impulse can only be estimated by palpation. On the introduction of the cardiograph, its results were looked forward to with the greatest interest, in the hope that they would throw much light upon this point. Any hopes, however, of assistance as regards estimating the energy of the heart from the cardiograph have proved fallacious, and its sole value lies in giving the record of the time of the cardiac movements, and, to a less extent, of their form.

The force of the impulse may be diminished by causes external to the heart. The presence of fluid or air in the pericardial sac, the existence of emphysema of the lungs, and sometimes even pneumothorax, pleurisy, and hydrothorax, may lessen the force of the impulse as felt by the hand. The

pulsation may also be enfeebled by causes acting upon the heart, such as nervous interference with its action, and general causes which affect its energy, as, for instance, in pyrexia, anæmia, and inanition. The force may further undergo changes as the result of local causes operative upon the organ itself, as when myocarditis results from pericarditis or endocarditis, and when cardiac failure occurs as the result of long-continued valvular disease.

The force of the pulsation may appear to be increased from extrinsic causes. In those who are thin the pulsation is more distinctly felt than in those who are fat, and in retraction of the left lung, from any cause whatsoever, there is not merely an increased area, but an exaggerated force of the pulsation.

Nervous interferences with the action of the heart give a more forcible pulsation, and muscular exertion at all times increases its energy. In the earlier stages of pericarditis and endocarditis the apex beat is almost invariably augmented in force, and, as the result of hypertrophy following valvular lesions, pericardial adhesions, renal and pulmonary affections, and other extrinsic causes of alteration, the force is increased.

*Systolic Recession.*—The recession or indrawing of the intercostal space around the apex beat has already been mentioned as regards inspection. This may be more distinctly appreciated on the application of the hand. On palpation in such a case the thrust of the apex beat is felt to be accompanied by a distinct drawing inwards of the parietes around it. This, however, is not all, for a distinct impulse of rebound or recoil is felt along with the diastole; and the existence of the systolic recession and diastolic recoil constitute the most characteristic symptoms of adherent pericardium.

*Rhythm of Impulse.*—By palpation the rhythm of the cardiac movements may be estimated with great accuracy. Simple irregularity in the succession, and inequality in the size, of the pulsations are extremely common symptoms in cardiac failure. Grouping of the pulsations may take place; the beats occurring in twos, or threes, or in larger numbers, giving the cardiac phenomena associated with the bigeminal pulse and its analogues.

A double impulse, consisting of a stronger pulsation followed by a weaker one, was described long ago by Skoda, and more recently by Leyden and Roy. It has been attributed by these observers to alteration in the systole of the heart, consisting in a contraction in which both ventricles participated, followed by another in which one alone took part. To this opinion it is perfectly impossible to subscribe. The two ventricles are so intimately united in structure by the interlacing of their fibres, that it is theoretically improbable that such an explanation as that which has been advanced by those observers can account for the appearances. It is, no doubt, true, that when the heart is dying some of its chambers persist in pulsating independently of the others; but, when this occurs, the form of the systole is entirely modified, and it is in the highest degree unlikely that for long periods the right ventricle should pulsate twice for one pulsation of the left. While fully admitting, therefore, that the left ventricle may give rise to pulsations of a character too feeble to produce a pulsation in the radial or other distant artery, it is impossible to allow that it does not pulsate at all; and, as has been shown by Mackenzie in some instances of this kind, a feeble pulsation may be made out in the carotid artery, while there is none in the radial.

*Accompaniments of Impulse.*—The contraction of the heart may be attended by certain accompaniments only to be appreciated by palpation. Such adventitious phenomena may be produced by causes outside the heart proper, or by its internal mechanism. It occasionally, but not very commonly, happens that, on applying the hand over the præcordia, friction may be felt. It gives a sensation of roughness exactly similar in character to that which is felt over a patch of dry pleurisy.

From the time of Senac it has been known that fluctuation may be felt over the heart, in copious pericardial effusion. In a patient whose case is recorded by this author, the fluctuation was felt during palpation of the heart, and its position was in the third, fourth, and fifth intercostal spaces.

The accompaniments which are found along with the



cardiac movements as the result of internal disorders are of the nature of vibrations or thrills. Corvisart was the first author who referred to this phenomenon, and he speaks of it as, "*Un bruissement particulier difficile à décrire.*" By Laennec the thrilling vibration was termed "*Frémissement cataire,*" in consequence of its resemblance to the sensation experienced by the hand on stroking a cat contentedly purring. Although this physical sign was first described as pathognomonic of mitral obstruction, the thrill may be produced at any of the cardiac orifices. At the aortic orifice both systolic and diastolic thrills are common, although the former, denoting obstruction, is much more frequent than the latter, indicating regurgitation. The systolic thrill is occasionally felt throughout the whole chest; the diastolic thrill is rarely felt except in the præcordial region, more especially towards the lower part of the sternum and in the neighbourhood of the apex beat.

Thrills produced at the mitral orifice are in the greatest proportion of cases of presystolic or diastolic rhythm, and denote obstruction at the left auriculo-ventricular orifice. They are always felt with their greatest intensity about the region of the apex and are not, as a general rule, propagated to any considerable distance from it. This rule is, nevertheless, subject to certain exceptions, and thrills of either rhythm may be transmitted over a considerable area of the parietes. Systolic mitral thrills are exceptional. When present, they probably depend upon a considerable degree of narrowing as well as of rigidity of the mitral orifice. Such systolic thrills may be conducted to a very considerable distance over the parietes, but their maximum intensity is about the apex.

Thrills originating at the pulmonary orifice may be systolic or diastolic. On account of the proximity of the pulmonary orifice to the surface, lesions of the cusps, whether obstructive or regurgitant, in most instances give rise to vibratile sensations; as pulmonary lesions are, however, far from common, such thrills cannot be regarded as frequent symptoms. They are necessarily much more common in congenital heart disease than in any other condition.

Tricuspid thrills are excessively rare. Even when there

is tricuspid obstruction, presystolic and diastolic thrills are seldom to be felt, while a systolic thrill accompanying regurgitation is practically unknown. As tricuspid incompetence is the commonest of all valvular lesions this fact is extremely significant.

Occasionally there is a systolic thrill accompanying the expansile pulsation over an aneurysm of the aorta or one of the greater arteries, and in cases in which the aorta is dilated and projects up to, or farther than, the summit of the manubrium sterni the thrill is not at all infrequently present. In those cases, already referred to, furnishing a systolic impulse in the second left intercostal space, produced by the pulmonary artery or conus arteriosus, the systolic impulse occasionally, although rarely, is accompanied by a thrill.

In the second left intercostal space there is a much more pronounced symptom of this kind in certain instances of congenital heart disease. When the ductus arteriosus is permanently patent a very distinct thrill is to be felt—a thrill which distinctly follows the systole of the heart, and persists until the diastolic phase has existed for some time. The reason for this is obvious, inasmuch as the blood stream flows from the higher pressure of the aorta to the lower pressure of the pulmonary artery. It must therefore generally occur after the aortic pressure has reached a certain level, and will persist until it has fallen, at least to some extent. In some of these cases the thrill is to be felt persisting throughout almost the entire cardiac cycle.

It might be expected that by means of the cardiograph a record of the vibrations constituting a thrill might be obtained. The tracings published by some observers give appearances somewhat like the movements which the lever might be supposed to give in consequence of vibratile impulse, as, for example, in the series of tracings in mitral obstruction obtained by Galabin. When these tracings, however, are carefully examined, it will be seen that the apparent vibrations are not sufficiently frequent to correspond to the thrill, and they are, therefore, probably produced by jerks communicated to the lever from slightly interrupted movements of the heart. No satisfactory result has ever attended any of my

own efforts in this direction, and the tracing (Fig. 24) which is annexed, obtained by means of the cardiograph with a rapidly revolving cylinder, from a patient with mitral obstruction



FIG. 24.—Tracing from the apex beat of a case of mitral obstruction with marked thrill.

tion giving rise to a most marked presystolic and diastolic thrill, shows absolutely no trace of the vibrations.

**PERCUSSION.**—It seems probable that the method of percussion was in vogue amongst the early Greek physicians, for, as Gee infers, it had apparently been established before the time of Celsus for the purpose of distinguishing between ascites and tympanites. It was never applied, however, to the diagnosis of chest affections until last century, when Auenbrugger produced that work which was the forerunner of all modern methods of physical examination.

Corvisart, who translated and annotated Auenbrugger's work nearly half a century after its appearance, earned undying recognition for calling attention to the importance of percussion and for extending its application, more particularly to the diagnosis of heart disease.

In the portions of his Treatise devoted to the heart Auenbrugger is by no means so luminous or so accurate as in the Observations referring to affections of the lungs. In the twelfth and thirteenth Observations he mentions the changes in the percussion sound produced by effusion into the pericardial sac, and by dilatation of the heart, linking such changes with other characteristic symptoms of these affections. Such are the earliest references to the method of percussion as applied to affections of the heart.

The great principle discovered by Auenbrugger is that all percussion sounds must be explained by reference to the

physical condition which is present. This principle appears incontrovertible. Nevertheless, after universal acceptance during a few years, heretical views were enunciated by Piorry, who attempted to found the new theory that every organ of the body yields its own characteristic sound on percussion. Skoda turned the minds of men back to the truth of Auenbrugger's position, very clearly expounded his principle, that the percussion sounds produced depend upon the physical conditions which underlie them, and demanded that all observations must be reconciled with the laws of sound.

The method of percussion practised by Auenbrugger was direct or immediate, that is to say, he tapped the chest directly with the tips of the fingers drawn together and stretched straight out, the chest being covered by a garment, or the hand by a glove, which was not to be of smooth leather. Corvisart followed the same method, and it was not until the investigations of Piorry that mediate or indirect percussion was substituted for immediate or direct. Piorry introduced a pleximeter made of ivory placed between the chest and the fingers used to percuss, and he describes that the fingers should be kept half bent so as to employ their tips—the method which is now universal. The plessor or percussion hammer was introduced by Wintrich; the employment of the pleximeter and plessor is attended by doubtful benefits.

Percussion as applied to the heart is necessarily of no value in regard to changes in its structure; it can only throw light upon its size and relations. There is therefore a twofold aim in applying percussion to the investigation of the heart. First, to determine the total space in two dimensions occupied by the heart, projected as it were upon an approximate plane; secondly, to ascertain how much of it is covered by lung tissue, and to ascertain the presence or absence of any abnormal structure in contact with the heart or great vessels. In mapping out the extent of the thorax occupied by the heart, the extent of the deep or relative dulness is the object to be investigated. For this purpose percussion must be carried out with considerable firmness, and in this way the whole of the lateral and superior boundaries of the heart may be ascertained with a precision which is almost absolutely



accurate. The results of percussion have been frequently verified by myself at post-mortem examinations. This has been done by delimiting the extent of the deep dulness, and introducing barbed wires vertically; these retain in the deeper regions of the chest the lines marked out before opening the thorax. On no occasion has it ever occurred to me to find that the real position was at any point more than one-eighth of an inch from that which had been marked out by percussion. It is therefore impossible to agree with Guttman when he says that the right edge of the heart cannot be defined. The inferior border of the heart, resting upon the liver, cannot be distinguished by percussion, inasmuch as the physical characteristics of the two viscera are such as to give rise to similar acoustic phenomena. The best method of ascertaining the total area occupied by the heart is to make out in the first place the upper limit of deep liver dulness in the right mammillary line, and thereafter to percuss from without inwards along the ribs and interspaces until the relative dulness at the cardiac margin becomes manifest. The same course is to be followed round the entire margin of the heart. The highest point at which the cardiac dulness may be determined is at the upper border of the third left intercostal cartilage close to the sternum. From this level it runs inclining slightly downwards across the sternum to the right, and curving more and more, comes to be about two inches from mid-sternum at the fourth right costal cartilage, beyond which, on account of the presence of the liver, it cannot be made out. From the highest point the left border runs outwards and downwards in a crescentic line, and at the level of the fourth rib it is between three and four inches from mid-sternum. The best method of ascertaining the boundary between the heart and the liver is to map out the entire areas occupied by the two organs, when a line drawn from the angle where the margins—ascertained by the deep dulness of the two viscera—meet, to the right of the sternum, is to be drawn to the point where the left end of the liver and the lower margin of the heart are found to be in contact.

The superficial or absolute dulness of the heart corresponds exactly with the portion of the right ventricle which is left

uncovered by the lungs. It is therefore somewhat triangular in form and may be regarded as having three sides, two of which only can be ascertained directly by means of percussion. The boundaries of this area vary in health with the respiratory movements. In quiet respiration or when the lungs are altogether at rest the right border of the superficial dulness of the heart is usually at mid-sternum. The left border begins at the upper border of the fourth costal cartilage, and curves outwards and downwards, crossing the fourth intercostal space and the fifth rib until it reaches the fifth intercostal space, at which point it curves slightly inwards, until it reaches the sixth rib, at which point the superficial cardiac and hepatic dulness merge. The average extent of the superficial dulness of the heart under such conditions may be stated by giving its vertical extent from the upper level of the fourth costal cartilage to the level of the sixth chondrosternal articulation as 2 inches, and its breadth at the level of the fifth costal cartilage as being  $1\frac{1}{2}$  inches. On forced inspiration and expiration the size of this area becomes respectively diminished and increased.

The size of the deep cardiac dulness is modified by all changes in the size of the heart. It therefore happens that in hypertrophy, and still more in dilatation, the area of cardiac dulness becomes enlarged in certain directions. In such affections of the right side of the heart the transverse extent of the dulness is chiefly enlarged, and in the case of similar changes on the left side of the heart the vertical extent is more particularly enlarged. Changes in the extent of cardiac dulness occur sometimes with a good deal of rapidity, especially in conditions of cardiac dilatation, probably on account of fluctuations in the quantity of the blood which the cavities of the heart contain.

In cases of considerable pericardial effusion alterations in the area of dulness are effected by changing the position of the patient. In the recumbent posture the dulness on percussion is somewhat smaller than when the patient sits upright, and the apex beat is more appreciable than in the sitting posture on account of the tendency of the fluid to gravitate backwards in the recumbent position and forwards in the erect posture.

In cases of effusion into the pericardial sac, the extent of the deep cardiac dulness is considerably increased, and it at the same time assumes a characteristic form which is fully described in the section dealing with such conditions.

The size of the area of superficial dulness is not so much affected by circulatory disturbances as by modifications in the condition of the respiratory organs. It nevertheless undergoes modifications in some affections of the heart. In cardiac hypertrophy and dilatation the area is often increased to some extent, and in pericarditis with considerable effusion this is even more conspicuously the case. Such alterations of the area of superficial cardiac dulness are small compared with the alterations which it undergoes in certain diseases of the lungs. In cases of collapse or retraction of either or both of the lungs the extent of superficial dulness may be enormously increased, while, on the other hand, in pulmonary emphysema it may be absolutely obliterated by the encroachment of the anterior borders of the lungs.

AUSCULTATION.—The causation of the heart sounds has already been fully discussed, and in the present section the clinical phenomena of health and disease alone require to be studied.

*Cardiac Areas.*—It need hardly be remarked that for convenience in clinical observation there are four conventional areas in which the characters of the sounds relating to the different orifices are ascertained.

The aortic area is situated at the second right chondro-sternal articulation, for the reason that the aorta is nearer the surface at this point than at any other, so that sounds generated at the aortic orifice are best conducted to this spot. Murmurs taking their origin in abnormal conditions of the aortic orifice and cusps are frequently, however, heard more distinctly at other points in the neighbourhood than in the aortic area itself, to which fact reference will be more fully made at a later stage. The mitral area is situated at the apex of the heart. This does not necessarily mean the visible or tangible apex beat, because in certain conditions, more particularly in dilatation or hypertrophy of the right ventricle, the apex beat is not produced by the apical part of the

heart. In any case of doubt the correct position of the cardiac apex must be determined by means of percussion, which clears up any doubt as regards its true position. At this point the left ventricle comes comparatively close to the parietes, and, therefore, serves as a means for the conduction of any sound vibrations arising at the mitral orifice. The pulmonary area occupies the inner end of the second left intercostal space, at which point the pulmonary orifice and its cusps are very near the surface. The tricuspid area may be regarded as having its centre at the fifth left chondrocostal articulation, within the limits of the superficial dulness of the heart, but compared with the other areas it is somewhat larger in size.

*Graphic Diagrams.*—Diagrammatic representations of the sounds of the heart and of their modifications, by means of conventional symbols, were first introduced by Gairdner. Many subsequent observers have extended and altered these graphic diagrams, but by no one has the subject been so thoroughly elaborated as by Wyllie.

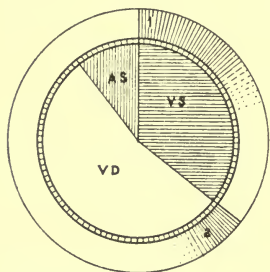


FIG. 25.—Diagrammatic representation of the cardiac cycle. The inner circle shows the movements of the heart: AS=auricular systole, VS = ventricular systole, and VD = ventricular diastole. The outer circle shows the normal sounds of the heart: 1 = first sound, and 2 = second sound. The narrow circle between them is divided into hundredths of seconds in order to have the opportunity of giving an accurate representation of the actual duration of each phase.

The movements and sounds of the heart may be represented as forming a circle, or they may be depicted as occupying different parts along the base line. In the accompanying illustration (Fig. 25) designed by me for a former work, a complete cardiac cycle is represented. In it the inner circle is subdivided so as to show the systole of the auricles, the systole of the ventricles, and the diastole of the ventricles, passing into the period of repose. It is surrounded by a narrow band subdivided so as to represent hundredths of a second, which renders it possible to represent accurately the average duration of the different phases of cardiac activity. Surrounding this

in turn is an outer circle upon which are represented the first, or long, and the second, or short, sound.



The cardiac sounds may be very conveniently represented, as in Fig. 26, by two rectangular outlines occupying spaces having a definite relation to the duration of the sounds.

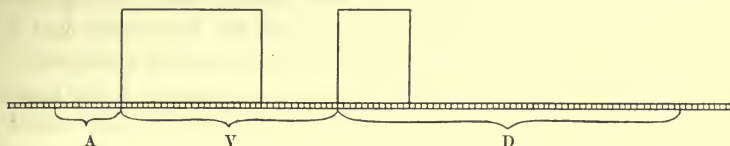


FIG. 26.—Normal heart sounds. A, auricular systole; V, ventricular systole; D, ventricular diastole.

By dividing the base of the line so as to represent hundredths of a second the relative size of these spaces is obtained.

*Intensity of Sounds.*—The relative intensity or loudness of the different cardiac sounds has already been referred to in a previous section, and it must be concluded from the facts therein mentioned that the loudness of the normal cardiac sounds, as heard through the chest-walls, does not, by any means, depend on their intrinsic intensity. It is conditioned, in great part, by the distance of the orifice at which they are produced from the surface, and by the nature of the media through which the vibrations have to pass. It will be remembered that from the observations of Vierordt, the first sound produced by the left ventricle is, at every age, louder than that produced at the right; while, up to middle life, the second sound in the pulmonary area is louder than that in the aortic.

It is extremely difficult, from a clinical point of view, to determine the relative loudness of the mitral and tricuspid first sounds. They are different in character, being somewhat lower in pitch and rather longer in duration in the mitral than in the tricuspid area. On the other hand, it is easy to determine that, in conditions of health, the pulmonary second sound is almost invariably of greater intensity, as well as higher pitch, than the corresponding sound in the aortic area. From the much higher pressure in the aorta than in the pulmonary artery, it might be expected that the aortic second sound would be louder than the pulmonary, and the fact that this is not so is probably to be explained by the deeper position of the aortic cusps, and also of the aorta itself, as compared with the pul-

monary artery. It may be objected to this that, for the same reason, the first sound in the tricuspid area should be more distinct than that in the mitral area. But it must be borne in mind that the thickness of lung intervening between the apex of the heart and the thoracic parietes is so thin that it can produce but little interference with the sound vibrations.

*Changes in Intensity.*—The various movements of the heart may be attended by modifications of the normal sounds which accompany the different phases of cardiac activity. These are twofold in character. There may, in the first place, be simple alterations in the intensity of the sounds, or changes in their rhythm. There may also be other phenomena, which have no counterpart under ordinary circumstances, and these may,

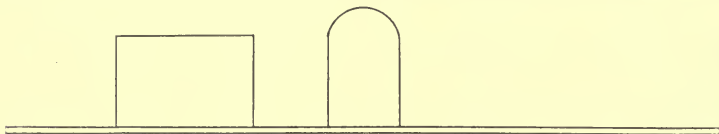


FIG. 27.—Accentuation of aortic second sound.

therefore, be termed new or adventitious sounds. Such sounds may accompany or replace the normal sounds of the heart.

Simple modifications of the intensity of the sounds of the heart are of frequent occurrence, and furnish indications as well for diagnosis as for treatment.

In considering such modifications it is advisable to consider, in the first place, the modifications of the second sound. An increase in the intensity, or, as it is commonly called, an accentuation, of the second sound may be produced at either of the arterial orifices. Normally, as has been shown, the pulmonary second sound is louder than the aortic, and if it should so happen that the sounds in both areas are of about equal intensity, it necessarily follows that the aortic sound is accentuated. It is not so easy to establish the fact of increased intensity of the pulmonary second sound; nevertheless, by dint of practice and experience it becomes by no means difficult to ascertain whether the pulmonary second sound is of greater intensity as compared with the aortic second sound. Accentuation of the aortic second sound depends on high arterial pressure, and it, therefore, is met with in many lesions

of the aorta, such as dilatation and aneurysm, as well as in changes in the peripheral arteries, more especially such as are summed up by the term arterio-sclerosis. It is extremely common in renal diseases. Accentuation of the second sound in the aortic area may be represented as shown in Fig. 27.

Accentuation of the pulmonary second sound may be produced by any increase of pressure within the pulmonary circuit,

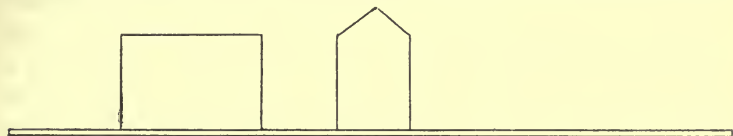


FIG. 28.—Accentuation of pulmonary second sound.

such as commonly occurs in pulmonary lesions obstructing the passage of blood through the lungs, *e.g.* emphysema or fibrosis, or from diseases of the orifices and valves of the left side of the heart, which produce consecutive changes in the pulmonary circulation. Accentuation of the pulmonary second sound may be represented as shown in Fig. 28.

Diminution in the intensity of the second sound occurs as a consequence of diminished blood pressure in the aorta or the pulmonary artery. The fall in pressure may have its origin in diminished access of blood to the ventricles, so that a smaller amount is ejected with the cardiac systole; to

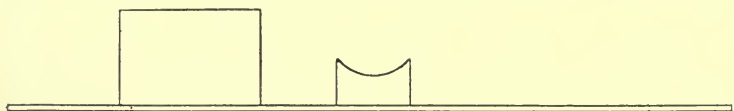


FIG. 29.—Diminution of aortic second sound.

diminished energy of the cardiac muscle; or to lessened resistance in the peripheral arteries.

The aortic second sound is diminished in intensity in such conditions as mitral obstruction, or lung disease, by which the return of blood to the left ventricle is impeded. It is also lessened in all affections which weaken the left ventricle, including such varying states as pyrexia on the one hand, and fatty degeneration on the other; and it is also reduced in loudness in conditions when the arterioles are relaxed.

Diminished intensity of the aortic second sound may be shown diagrammatically as in Fig. 29.

Diminished intensity of the pulmonary second sound is far from common. It might be expected to occur as a consequence of tricuspid incompetence, but this, as a matter of fact, can rarely be determined. Diminished intensity in consequence of an easier passage of the blood from the pulmonary blood vessels does not present itself as a common experience, and an absolute diminution in the intensity of the second sound, therefore, takes its origin almost always in a weakening of the walls of the right ventricle. A diminution of intensity of the pulmonary second sound, therefore, is to be



FIG. 30.—Diminution of pulmonary second sound.

regarded as a symptom of the gravest prognostic character. It may be represented as in Fig. 30.

Alterations in the intensity of the first sound may be brought about by modifications of any of the factors which have been referred to as engaged in maintaining blood pressure. Considerable variations in the character and loudness of the first sound are observed in health. In the thin and nervous, the first sound is apt to be high in pitch, ringing in quality, and short in duration; in the strong and vigorous, it is low in pitch, rumbling in character, and long in duration; while, in the fat and indolent, it is often so faint and short as to present a great contrast to the second sound. Such differences in the first sound are common to both sides, but are more particularly met with in relation to the first sound of the left ventricle as heard in the mitral area.

Increased intensity of the first sound produced by the left ventricle does not by any means always occur under the circumstances which might be expected to produce it. In hypertrophy of the left ventricle, associated with arterial degeneration and renal cirrhosis, it might be expected that the first sound would be much louder than in health. Such



an expectation, if formed, would fail of realisation. In these circumstances the first sound in the mitral area is often somewhat muffled in its character, and gives the impression of a long, sustained, low pitched sound. Under opposite conditions, when there is some dilatation of the left ventricle, the sound may often be loud in its intensity and high in its pitch. In certain stages of febrile conditions, when there is diminished

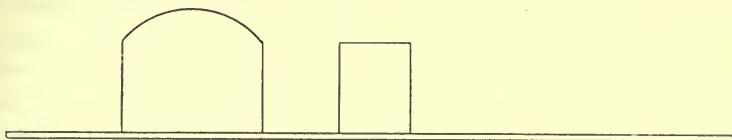


FIG. 31.—Accentuation of mitral first sound.

arterial pressure along with considerable excitement of the heart, the mitral first sound may again be heard of considerable intensity, as well as high pitch. Of cardiac lesions affecting the mitral first sound, it may be said, that in pure mitral obstruction the first sound, whether following the presystolic murmur or not, is often louder than in health, while in mitral incompetence the opposite condition prevails. Aortic changes tend to produce the low, rumbling, sustained first sound which has been alluded to as common in hypertrophy. Accentuation may be shown as in Fig. 31.

Diminished intensity of the mitral first sound takes

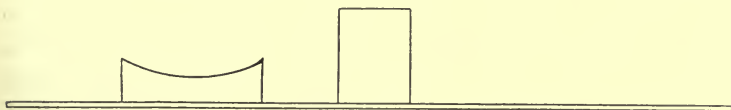


FIG. 32.—Diminution of mitral first sound.

place in the later stages of almost all pyrexial conditions, and speaks for weakness of the walls of the heart. When the weakening of the sound is so considerable as to render it almost inaudible, the symptom must be regarded as of the gravest significance. Of valvular lesions the only one which directly affects the first sound in the mitral area is mitral incompetence, in which the first sound, when not quite obscured by a murmur, is almost invariably weakened,

in spite of the frequent occurrence of some hypertrophy. In all conditions of degeneration of the walls of the left ventricle, the first sound undergoes diminution, and in advanced fatty degeneration it may be absolutely imperceptible. It is shown in Fig. 32.

The first sound over the right ventricle undergoes even greater variations than that produced by the left ventricle, as well in health as in disease. This, however, is more remarkable as regards diminution in its intensity; an increase in the loudness of the first sound in the tricuspid area is not such a marked feature as on the opposite side. True, when there is any obstruction to the passage of the blood into the left chambers of the heart, or to its onward course through the lungs, there is, along with accentuation of the pulmonary second sound, some increase in the intensity of the tricuspid

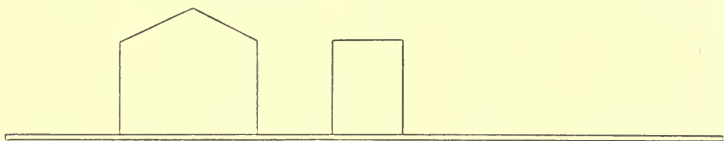


FIG. 33.—Accentuation of tricuspid first sound.

first sound; but when this goes beyond a slight limit the safety valve function is brought into action, and tricuspid regurgitation diminishes the intensity of the first sound. It, however, often happens that, with the accentuated pulmonary second sound, the first sound in the tricuspid area may be faint, or it may even be replaced by a murmur. Aortic and mitral lesions bring about such events; regurgitation in both instances being more likely to do so than obstruction; while, amongst diseases of the valves, those which affect the distribution of the pulmonary artery produce the greatest results, more especially such changes as induce emphysema, leading to great destruction of the capillaries of the lungs. Any disease, however, which causes deficient oxygenation of the blood, produces an interference with the passage of the blood through the pulmonary capillaries, and, in this way, tells back upon the right ventricle. Fig. 33 represents the condition.

Diminished intensity of the first sound in the tricuspid

area is found in all conditions involving weakness of the ventricular wall, such as the later stages of pyrexia, and all forms of mal-nutrition; while, in degenerative changes of the cardiac muscle, the tricuspid first sound may almost entirely fail. It is shown in Fig. 34.

A ringing or metallic quality of the heart sounds is found in pneumo-pericardium. This acoustic phenomenon is very often obscured by the splashing and churning accompaniments almost invariably present in this rare condition, and, as will be found below, in the only instance which has ever presented itself for my observation, the external accompaniments produced by the movements of the air and fluid in the pericardial sac almost entirely obliterated the character of the heart sounds.

*Changes in Rhythm.*—Alterations in the rhythm of the

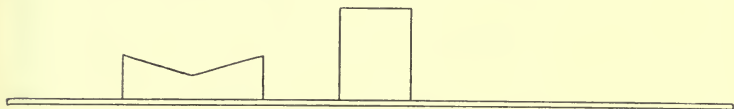


FIG. 34.—Diminution of tricuspid first sound.

heart sounds may occur in one, or other, or both, of two different ways. There may be interference with the relations of the first and second sounds to each other, or with the succession of the entire events of the cardiac cycle.

Of changes affecting the relations of the two sounds to each other, the simplest are those involving an alteration in the relative time occupied by the sounds and silences. As was previously shown in the chapter on physiological considerations, alterations in the rate of the heart beat are chiefly effected through modifications in the duration of the period of repose. As might be expected, therefore, when the heart is accelerated the pause which follows the second sound becomes shortened, and in any considerable acceleration the first and second sounds may become equidistant. Broadbent is of opinion that the lengthening of the systole produces such a prolongation of the first sound as to bring about equidistance of the first and second sounds. This, however, from long and careful observation, would appear to me to be an occur-

rence of the greatest rarity. Even in protracted high arterial pressure, associated with kidney disease, such a lengthening of the first sound is only exceptionally observed.

Doubling of the second sound, originally discovered by Bouillaud, has usually been ascribed to asynchronous closure, or tension, of the aortic and pulmonary valves. When we inquire more closely, however, into the views held by different observers as to the means by which this failure to act simultaneously is produced, we find considerable divergence of opinion. By von Bamberger it was attributed to an irregular contraction of the arterial walls, causing the propulsion of successive blood waves towards the sigmoid valves. Potain appears to have been the first to assume a difference of pressure within the aorta and pulmonary arteries as a cause of the asynchronous action of the valves, and his interpretation has, with various modifications, remained, to some extent, valid since its inception. It is perfectly true that Guttmann speaks of asynchronous closure of the individual cusps of one or other of the arterial valves, aortic or pulmonary, and that Sansom regards the second part of the doubling of the sound as being due to the sudden tension of abnormal mitral curtains, consequent upon the relaxation of the left ventricle. The opinion of Guttmann must be dismissed as in the highest degree improbable, if not, indeed, absurd, while Sansom's explanation, even if sufficient to explain the doubling of the second sound in cases of mitral disease, would not account for the physiological doubling.

Doubling of the second sound occurs under physiological conditions at the end of inspiration. In pathological circumstances it may have its origin in any condition which disturbs the normal balance of the circulation. Any interference with the pulmonary circulation, whether arising in the lungs or at the left side of the heart, may produce it, and any disturbance of systemic circulation giving rise to modifications of the pressure will also bring it about. It is in this way that arterial sclerosis and chronic disease of the kidneys produce doubling.

In truth, the mere existence of the doubling caused by physiological acts, such as holding the breath after a deep in-



spiration, above referred to, is sufficient to prove that slight alterations of the relative pressure in the different parts of the central apparatus are sufficient to produce a double second sound. And if double second sound can be produced by slight changes of relative pressure, it is unnecessary to go further in search of a cause for the persistent double second sound of mitral disease. The manner in which changes of relative pressure effect the result must, as is generally recognised, be by causing

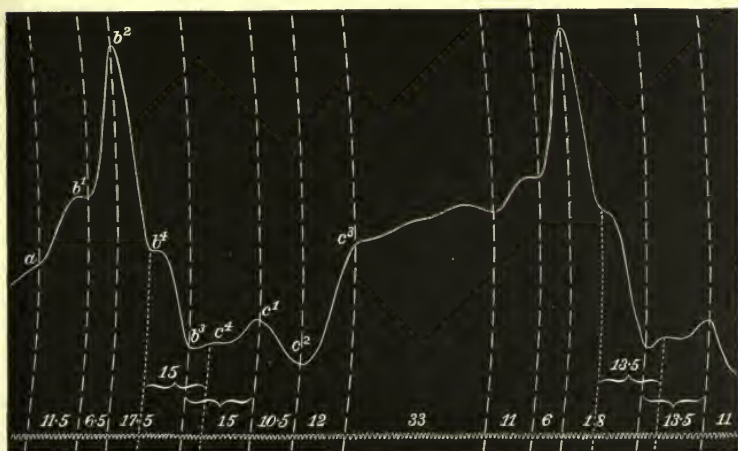


FIG. 35.—Tracing from conus arteriosus.

the systole of one ventricle to terminate a little later than that of the other.

Some years ago in analysing the tracing from the conus arteriosus which is annexed, an opportunity was seized by me to point out that the shoulder,  $b^4$ , upon the descending curve produced by ventricular contraction, was separated from the base,  $b^3$ , by the same interval of time as that which elapsed between two impulses,  $c^4$  and  $c^1$ , according with the second sound, and, it seemed to me, that in this tracing there was distinct proof that one of the ventricles ceased its contraction at the point,  $b^4$ , which bore the same relation to  $c^4$ , as  $b^3$  does to  $c^1$ . On which side this early cessation of systole occurred could not, as may be expected, be determined.

Discussion has taken place as to which of the two second sounds, aortic or pulmonary, takes place first in order of time. One opinion, strongly urged by Balfour, assumes that the closure of the sigmoid valves is accelerated on the side of the greatest pressure. This opinion has always appeared to me to charge nature with a defective provision for meeting excessive stress, and it flies in the face of our modern views as to compensation in cardiac disease. We know, as a matter of fact, how ample the provision is, and Cohnheim has eloquently enlarged upon the impossibility of placing a limit upon it.

The other opinion—stoutly asserted by Barr, and adopted by most of our recent writers—holds that the second sound is later on the side which meets most pressure, causing the ventricle therefore to be longer in overcoming the obstacle. This view is strictly in accord with what we know to be the inherent tendency of the cardiac mechanism, and it is also supported by clinical observation; for on careful auscultation of the aortic and pulmonary areas, in those cases of mitral disease which present the double second sound, it is possible in every instance to determine that the first of the two sounds has its greatest intensity in the aortic, and the second, which is the louder of the two, in the pulmonary area. This is an absolute demonstration that the second sound produced by the sigmoid valves at the pulmonary artery is later than that arising from the action of the aortic cusps. In other words, the right ventricle takes an appreciably longer time than the left to terminate its systole.

An apparent doubling of the second sound is at times to be heard at the apex of the heart, while it is absolutely inaudible in the aortic and pulmonary areas. This phenomenon must be carefully distinguished from the real doubling, which is always heard most distinctly at the base of the heart. So far as is known to me, this apparent doubling of the second sound is only heard in mitral obstruction. Its explanation is undeniably difficult.

It has been explained by Sansom in the following way. As the tension of the mitral cusps undergoes a change early during the diastole when the ventricle is relaxed after its contraction, the blood, kept under considerable pressure in the

left auricle, enters the ventricular cavity with some force. Passing to the ventricular side of the curtains of the mitral valve, this causes them to bulge, and thus produces a sound of valvular tension, which, following soon after the second sound, produces what seems very like a doubling of the sound. Boyd, who has recently written a most interesting paper upon this subject, is of opinion that the doubling when heard at the apex and not at the base, is an apparent, not an actual, doubling of the sound, and believes that the second element is produced at the mitral orifice. He points out that the phenomenon not infrequently passes into a distinct diastolic murmur. Doubling of the second sound is shown in Fig. 36.

Doubling of the first sound was regarded by Guttmann as due to asynchronous tension of the individual segments of the

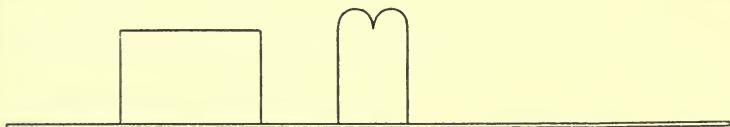


FIG. 36.—Double second sound.

auriculo-ventricular valves, consequent upon lack of uniformity in the contraction of the papillary muscles. It was considered by Hayden to be due to the resolution of the first sound into its two constituent elements, ventricular impulse and valvular tension. The former element of the doubling of the sound would therefore be expected to have a dull and muffled character, while the latter should be sharp and clear. Such, according to Hayden's statement, is actually the case in his experience. By D'Espine the doubling of the sound was regarded as caused by mitral vibrations, the mechanism of which he could only regard as obscure. Johnson regarded the first part of the doubling of the first sound as being produced by auricular systole in consequence of dilatation, and, more particularly, hypertrophy of the left auricle, whose contraction he believed to become sonorous. Bramwell accepts want of simultaneous action of the mitral and tricuspid valves, and he supposes that the doubling may be produced by such acceleration or retardation of the action of either ventricle as

will allow the first sound produced by it to be separated by an interval sufficient to be appreciated by the ear from the sound produced by the other ventricle. He regards the acceleration or retardation in the contraction of either ventricle as due to differences in the pressure of the blood in the two ventricles, by means of which the muscular fibres of the one are stimulated to contract before those of the other; or to alterations of the nerve apparatus of one ventricle, whereby the motor ganglia of one side are more or less irritable than those of the other; or to structural alterations in the muscular fibres of one vessel by which its contraction is more or less quickly affected than that of the other.

Sansom draws a distinction between real and simulated doublings of the sound, and points out, very justly, that there may not be an absolute and complete repetition of the systole of each ventricle, so that the entire process of emptying the one must succeed that of the other, but that it would be sufficient if the maximum tension occurred in the one at a sufficient distance of time from that of the other for the ear to distinguish the difference in time. He believes, however, that, at least in some cases, the apparent doubling of the sound is in part due to a presystolic tension of the mitral cusps produced by their being floated upwards during auricular systole. This is a relic of the exploded idea of the action of these cusps. He, therefore, concludes that the doubling of the first sound is due, not to a want of synchronism in the action of the ventricles, by means of which the mitral and tricuspid valves are brought into a state of maximum tension at different periods of the systole, but to a sudden shock communicated to the ventricular contents just before the occurrence of the systole.

Kriege and Schmall, as the result of cardiographic investigations, have come to the conclusion that in doubling of the first sound the first portion is produced by auricular, and the second by ventricular, systole. This cannot be accepted.

Doubling of the first sound is by no means so common as is the case with the second sound. The reason for this is not far to seek, and it has been put most cogently by Barr. The explanation is that the first sound has a considerable duration, and therefore the maximum intensity is spread over a some-



what wide interval. Doubling of the first sound can never really be a true doubling; otherwise almost the entire cardiac systole would be occupied by two alternate first sounds, which is absurd. It therefore follows that doubling of the first sound is an apparent, rather than a real, phenomenon, and all that takes place is that the maximum intensity of the left first sound does not coincide with the maximum intensity of the right first sound. It is, therefore, produced by some delay in the process of contraction of the two sides of the heart. The symptom is found in cases of chronic renal disease, with vascular and cardiac accompaniments; in obstructive lesions

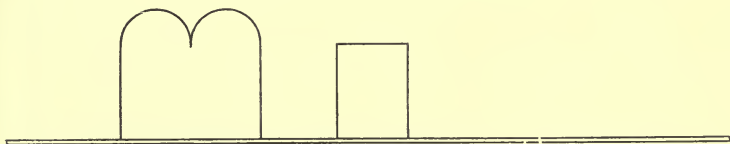


FIG. 37.—Double first sound.

of the lungs, and in degenerative conditions of the cardiac muscle. Double first sound is shown in Fig. 37.

*Adventitious Sounds.*—So far, simple changes in the characters and rhythm of the normal sounds have been considered. It is now necessary to deal with auscultatory phenomena which have no place in health.

*Sounds produced Externally.*—Among the acoustic phenomena which have no place in health are certain which take their origin in causes lying outside the heart altogether. These are, for the most part, produced within the pericardial sac. When there is any roughness of the pericardium so that its two surfaces grate harshly against each other, instead of gliding smoothly as in ordinary circumstances, a distinct friction sound is heard over the præcordia. This phenomenon, heard indeed by Laennec but misunderstood by him, and independently discovered and explained by his pupil Collin, varies widely in its characters, but has, nevertheless, certain well-marked and constant features.

One of the most characteristic points is that it is not conducted to any distance. It can only, in short, be heard over the region in which it takes its origin. It may accom-

pany every phase of the cardiac cycle—that is to say, it may be heard with the movements of the auricles or of the ventricles. It is usually heard along with ventricular systole and diastole; less commonly along with auricular systole, ventricular systole, and ventricular diastole; while it is rare to hear it accompanying one phase alone. In its character it is always more or less rough, with a creaking or grating quality. In addition to these various features it always gives the observer an idea of superficiality, and this is further borne out by the fact that a little additional pressure upon the stethoscope invariably

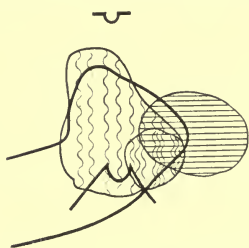


FIG. 38.—Tracing of chest in pericarditis with mitral incompetence. The thick lines show the upper and lower costal limits and the area of cardiac and hepatic dullness. The horizontal shading gives the area over which the mitral murmur was audible. The wavy lines show the area of friction, triple in rhythm at the apex—double elsewhere.

causes an increase in the intensity of the noise. Certain other properties possessed by the noise of pericardial friction are fully dealt with in the section describing pericarditis. Pericardial friction may be produced by any condition rendering the serous membrane rough, and, possibly, a condition of dryness may be sufficient to give rise to it. Besides simple pericarditis, the presence of tubercular, gummatous, and cancerous deposits may induce it. The existence of milk spots on the visceral pericardium, and even inequalities arising from arterial sclerosis, may, as has been shown by myself, give rise to the

sound of friction. The distribution of the friction murmur in an illustrative case is shown in Fig. 38.

Other more complicated noises may arise within the pericardial sac. When gas and fluid are present together, an extraordinary assemblage of noises is produced, which have, in greater or lesser degree, the qualities of sounds produced by churns or water-wheels. The splashing character of all these sounds will be more fully described in connection with pneumo-pericardium.

One extrinsic phenomenon connected with the lungs may be referred to in passing: it is the interrupted or cog-wheel respiration. This form of breathing is very commonly ascribed

to a condition of deficient elasticity of the lungs, and it is often held to be a precursor of tubercular deposition. For such an idea there is often no warrant. In many instances where the heart is hypertrophied, interrupted respiration may be heard throughout the entire chest, with perfect health of lung tissue. One of the most beautiful examples of this which has ever come under my notice is in the person of an athletic clergyman who pulled the fifth oar in the boat of his university several years ago at the annual Oxford and Cambridge race. The phenomenon has been present to my certain knowledge for five or six years, and it simply results from the action of a powerful yet healthy heart, surrounded by lung tissue of absolute integrity.

It is unnecessary in this place to refer to the cardio-pulmonary murmurs described by Potain, the mechanism of which, as suggested by him, it is impossible to accept. They have recently been thoroughly analysed in this country by Gordon Sanders, and will be discussed in the section describing the effects of relaxation of the cardiac walls.

*Sounds produced Internally.*—By the movements of the blood a large series of abnormal acoustic phenomena are generated in the heart and vessels. These are termed murmurs, in order to distinguish them from the normal phenomena heard in the heart and vessels, to which the name sounds is restricted.

*Cause of Murmurs.*—The production of murmurs, whether in the heart or in the blood vessels, has been the subject of a very large number of observations. Laennec at first ascribed the production of a murmur to a condition of over-distension of the heart; but, at a later period, as may be seen in Forbes's translation, he says:—"In the first edition of this work, I considered the bellows-sound of the heart as a sign of contraction of the orifices. No doubt it exists almost always in this case; but since the first publication of my treatise, I have very frequently met with it in individuals who had no lesion of any sort; while, on the other hand, I have seen ossifications of the valves which were not attended by this sound. I have likewise frequently observed it in the last agony, and in other circumstances when the heart is too full of blood, in which

latter case it sometimes quickly yielded to bloodletting. I formerly also was inclined to consider this phenomenon as connected with the redness of the inner coat of the arteries, considered by some modern writers as an inflammatory affection; but I have since then found the arteries quite pale and perfectly sound, in every case which I have had occasion to examine. In like manner, I can state with certainty, that the bellows-sound of the heart is very often met with when this organ is perfectly healthy. From these data it results that this phenomenon is attributable either to an organic or vital condition of the artery—a sort of spasm or tension—or else to a particular condition of the blood itself, or to the manner in which it is moved. The last supposition is inadmissible, inasmuch as the phenomenon exists sometimes in one artery only.”

The first satisfactory explanation of the production of murmurs came from Corrigan. Although this author allows what cannot now be granted, that murmurs may have their origin in some functional disturbances that need not at present be referred to, he undoubtedly discovered the real cause of murmurs. He showed that when any constriction was present in a channel through which fluid was flowing, a small stream passed from a narrow orifice into a wider tube, and when pursuing its way through the surrounding fluid gave rise to a rushing of the fluid with a trembling of the vessel, so that vibration was caused and a murmur heard. Gendrin attributed murmurs to friction between the blood and the interior of the vessel through which it is flowing, and this view was accepted by many observers. Chauveau may be said to have given the death-blow to the conception that simple friction could originate murmurs, and to have placed the teaching of Corrigan upon a definite basis. He showed by experiments on the carotid artery of the horse, that roughening of the interior of an artery does not cause murmurs, while the presence of a dilatation in any artery gives rise to them. He also observed, by auscultating the cervical vessels of a horse before and after repeated bleedings, that murmurs are not produced by changes in the quality of the blood. Taking advantage of the investigations of Savart, he concluded that murmurs are produced



by the vibrations of a fluid vein within the vessels; this fluid vein he believed always to occur on the entrance of the blood into an absolutely or relatively dilated part.

Heynsius pointed out that murmurs had their origin primarily in the fluid, and that friction between the fluid and the wall of the vessel containing it could not possibly be a cause. He further showed that the sound was produced in a wider part of the vessel, allowing of eddies in the fluid which produced the murmur.

Murmurs are primarily vibrations produced in the blood itself. Whether the theory of the fluid vein or the theory of eddies in the fluid has the greater weight of evidence in its favour need not here be discussed; suffice it to say that probably both views are true, and that in some instances the former is most applicable, while in others the latter is the one most likely to occur. When the blood passes through a small opening into a wide space beyond, the fluid vein is that which commends itself most to the judgment. When, on the other hand, the lumen is somewhat narrowed at one point, the theory of the eddy beyond seems most worthy of acceptance. That we are perfectly acquainted with all the physical conditions that can give origin to cardiac or vascular murmurs is a view that cannot for a moment be entertained, and the philosophical opinion of Fagge is worthy of being carefully pondered.

*Rhythm of Murmurs.*—Murmurs may be produced at any part of the cardiac systole during which the blood is flowing. It therefore follows that they may occur during the systole of the auricles. Murmurs occurring during this period of the cardiac systole are commonly termed presystolic, inasmuch as they occur before the obvious contraction of the ventricles. This term was erroneously attributed to Gendrin by Fauvel, who first described murmurs of this rhythm. They were termed by Gairdner auricular systolic or A. V. murmurs, and this terminology is usually adopted in the Glasgow

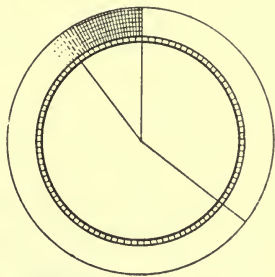


FIG. 39.—Presystolic murmur. In this and the five similar diagrams a murmur is distinguished from a sound by the double shading.

Medical School. Such murmurs can only occur at the auriculo-ventricular orifices, and, as the blood during the auricular systole flows from the auricle into the ventricle, they must denote an obstruction at those orifices. They may be represented as in the diagrams, Fig. 39 and Fig. 40.

The mode of production of these murmurs has given rise to much controversy. Originally discovered and clearly described by Fauvel, they were brought into prominence by the writings of Gairdner, whose lucid descriptions placed their causes and characters fully before the scientific world. Ormerod, not long after the appearance of Gairdner's work, assailed the views enumerated by Fauvel and expounded by him chiefly on the ground that the auricle is not sufficiently strong to produce such a murmur as the presystolic. Barclay and Turner somewhat later supported a view similar to that suggested by Ormerod, and more recently Dickinson and M'Vail

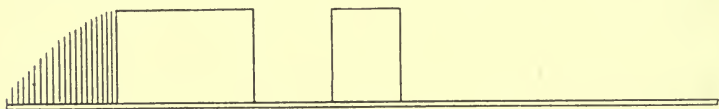


FIG. 40.—Presystolic murmur.

independently arrived at the same conclusions. While these sheets are going through the press Brockbank has in a very ingenious paper espoused the same opinion, somewhat differently expressed, and departing from these authors in some points of detail. According to all these observers the murmur is produced by the systole of the ventricle. It is held by them to begin at an earlier period than usual, and to be continued until closure of the valves sets in. In one of the most recent of the important works on medicine Tripier and Devic actually term the murmur protosystolic. These views have been stoutly combated by many authors, but more particularly by Balfour and Fagge.

That the explanation of Fauvel and Gairdner is correct cannot, in my opinion, be doubted for one moment. Two facts seem to me absolutely destructive of the hypothesis of Ormerod and his followers. There is, first, the important consideration that the murmur begins in many instances with

the second, and continues without intermission until the first sound. This in itself is fatal to such views. The unique case of obstruction of the tricuspid orifice narrated by Gairdner is, secondly, overwhelming evidence in favour of what may be termed the orthodox view. In this interesting case a tumour projected from the wall of the right auricle. It could not interfere with the closure of the tricuspid valve, but during auricular systole it came down over the cusps and produced a loud presystolic murmur.

This subject will be again referred to in connection with the special details of mitral and tricuspid obstruction.

Murmurs may be produced during the ventricular systole, and are hence usually termed systolic; but, for purposes of medical accuracy, Gairdner terms such murmurs ventricular systolic or V. S. murmurs. As during the ventricular systole the blood in abnormal conditions may pass through any of the four great cardiac orifices, it follows that a murmur of this rhythm may be produced at any one of these. At the auriculo-ventricular orifices, which ought to be closed during the systole of the ventricles, the systolic murmur denotes regurgitation, while, at the arterial orifices, which ought to be widely opened during this phase, the murmur implies the presence of some obstacle to the exit of the blood, and it is therefore obstructive.

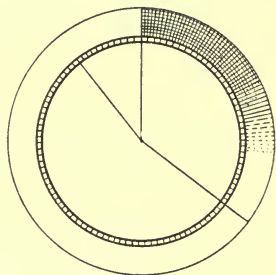


FIG. 41.—Systolic murmur.

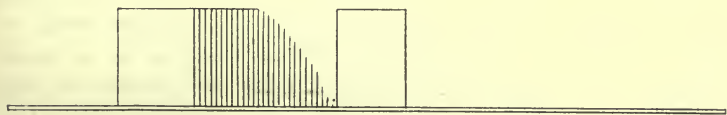


FIG. 42.—Systolic murmur following first sound.

well as in Fig. 42 and Fig. 43, where the murmur follows, in Fig. 44, where it accompanies, and in Fig. 45, where it altogether replaces the first sound

The diastole and period of repose may also be accompanied by murmurs which are usually termed diastolic, or, accord-

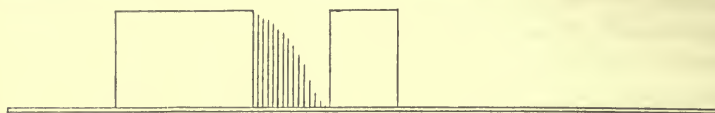


FIG. 43.—Systolic murmur following first sound.

ing to Gairdner, ventricular diastolic or V. D. murmurs. These may occur at any of the four orifices, seeing that in

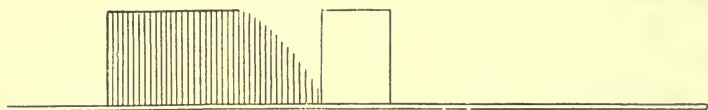


FIG. 44.—Systolic murmur accompanying first sound.

abnormal circumstances it is possible for the blood to be in motion through any one of them during this phase of the cardiac cycle. Taking up the arterial orifices first, for the sake

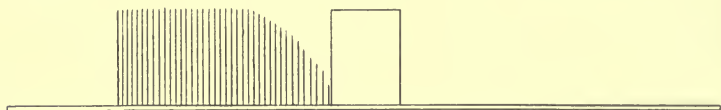


FIG. 45.—Systolic murmur replacing first sound.

of simplicity, the diastolic murmur means that an escape is permitted backwards from the artery into the ventricle, and

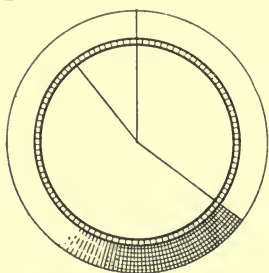


FIG. 46.—Diastolic murmur.

such a murmur therefore denotes regurgitation of the blood from incompetence of the valves. At the auriculo-ventricular orifices such a murmur means that there is some interference with the passage of the blood from the auricle into the ventricle, and therefore denotes obstruction. Such murmurs may follow, accompany, or replace the second sound. Murmurs occurring during this phase of the cardiac cycle at the auriculo-ventricular orifices frequently do not



coincide with the diastole but occur at a somewhat later period, and it is common to term them "post-diastolic."



FIG. 47.—Diastolic murmur following the second sound.

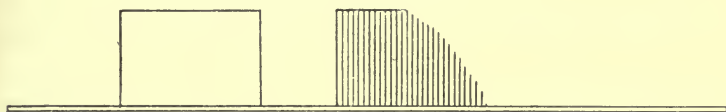


FIG. 48.—Diastolic murmur accompanying the second sound.



FIG. 49.—Diastolic murmur replacing the second sound.

Their rhythm may be represented by the diagrams of early and late diastolic murmurs, in Figs. 46, 47, 48, 49, and 50.

One peculiar rhythm of murmur deserves mention here—

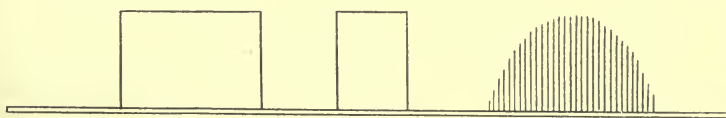


FIG. 50.—Late diastolic murmur.

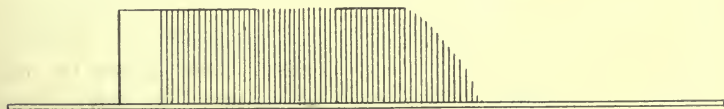


FIG. 51.—Continuous systolic and diastolic murmur in patent ductus arteriosus.

that which occurs in, and will be more especially described under, a patent condition of the arterial duct. It begins as a late systolic murmur, and is continued beyond the second sound, as shown in Fig. 51.

Many combinations of these murmurs may be found, those belonging to any two, or all three, of the different phases being associated. In Fig. 52 presystolic and systolic murmurs are represented as associated together; in Figs. 53 and 55, systolic and diastolic; in Fig. 57, diastolic and presystolic; and in Figs. 54 and 56 all three co-exist.

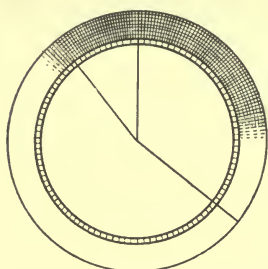


FIG. 52.—Presystolic and systolic murmurs coexisting.

*Character of Murmurs.*—Cardiac murmurs present many different varieties of character. Soft blowing murmurs occur most frequently. Such murmurs are, as a general rule, more commonly found in regurgitation at an orifice, but this is far from being an absolute rule. Rough, grating murmurs are more particularly found in obstruction of orifices, but regurgitant murmurs sometimes possess such qualities. Less frequently a murmur presents such a whistling or singing character as to merit the term musical murmur. The lesion which of all others is most likely to give rise to such a phenomenon is the diastolic aortic murmur.

*Character of Murmurs.*—Cardiac murmurs present many different varieties of character. Soft blowing murmurs occur most frequently. Such

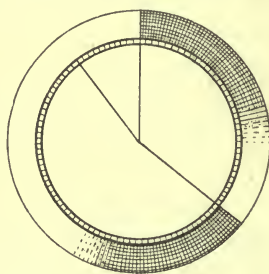


FIG. 53.—Systolic and diastolic murmurs coexisting.

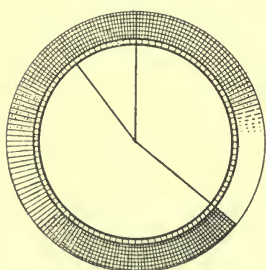


FIG. 54.—Presystolic, systolic, and diastolic murmurs coexisting.

The reasons for these differences are difficult to ascertain. Some of the work of Bergeon was intended to elucidate problems of this kind, and the same end was aimed at by Barié in more recent observations on the subject. It cannot be held, however, that their researches, ingenious though they were, have in any appreciable degree cleared up the physical conditions underlying the varying character of cardiac murmurs. It is no doubt a subject of the greatest difficulty, seeing that so many factors

are concerned in the production of these murmurs—the energy of the heart; the pressure of the blood; the size and form of the orifice; the extent and form, as well as the character and structure, of the space beyond it—these, and possibly other factors still, must be concerned in impressing

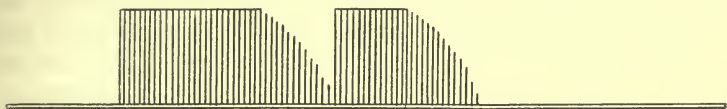


FIG. 55.—Systolic and diastolic murmurs.

particular characters upon murmurs; and modifications in any or all of them may produce great variation in their qualities.

The character of murmurs cannot be held to have much diagnostic significance except in the case of the rough and rumbling murmurs of obstruction at the mitral and tricuspid

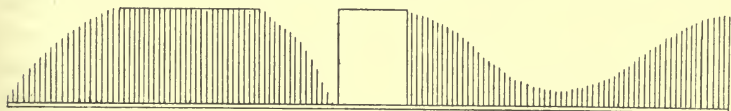


FIG. 56.—Presystolic, systolic, and diastolic murmurs in combination.

orifices. It must be admitted that the quality of these murmurs is almost, if not quite, pathognomonic.

The intensity or loudness of murmurs is also of extreme variability. Murmurs of the same rhythm and position may, in two different individuals, present a great contrast, being,

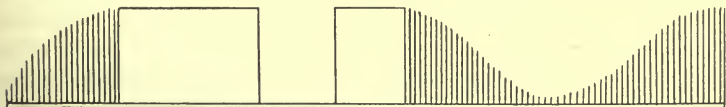


FIG. 57.—Presystolic and diastolic murmurs in combination.

for example, in one case soft and blowing, and in another harsh and grating. This variability is not only present when the symptoms in two different cases are compared, it is often manifested in the same individual when auscultated at different times. That murmurs were liable to such fluctuations in intensity has been long known, and Sanders more particularly directed attention to this important subject.

The loudness of a murmur has no direct ratio to the gravity of the lesion by which it is produced. If there be any relation at all, it is that the seriousness of the affection presents an inverse ratio to the loudness of the murmur. This statement seems paradoxical, yet it is approximately true. The most serious valvular lesions are those in which, on account of failure of the muscular walls, no murmur at all can be produced; and, as will be mentioned several times in the sequel, patients brought into hospital in a moribund condition, with no cardiac murmurs, develop these under the influence of absolute rest and appropriate remedies. The appearance, therefore, of a murmur is often to be regarded as the most hopeful fact about a patient, seeing that it is a precursor of returning cardiac energy.

The position of the patient has considerable influence on the intensity of cardiac murmurs. Obstructive murmurs generated at the auriculo-ventricular orifices are, as a general rule, of greater intensity in the sitting or standing posture, and they become feebler, or even disappear altogether, when the patient lies down. The opposite is the case with regurgitant murmurs at these orifices, for they are, as a rule, more distinct in the recumbent position. This fact was well known to Stokes, and is fully described in his great work upon the heart. It is, indeed, so well known, that it is almost a matter of ordinary routine, if there be any suspicion of disturbance at the great venous orifices, to make the patient lie down and auscultate the mitral and tricuspid areas in the recumbent posture. Tricuspid systolic murmurs particularly, including the systolic murmur over the conus arteriosus, are often brought out in this way when they are entirely absent in any other position. It is, therefore, extremely difficult to understand how Guttmann can have committed himself to the unwarranted statement that all murmurs are louder when the patient stands or sits than in the recumbent position, and that in the latter position the softer murmurs are sometimes entirely suppressed. Aortic and pulmonary murmurs are not subject to such variation on change of posture. They are altogether more stable. The reason for such fluctuations in intensity is probably to be sought for in the effects produced by



gravitation. The murmurs of obstruction at the auriculo-ventricular orifices, produced either by the contraction of the auricles, the aspiratory action of the ventricles, or the inertia of the onward current of the blood, will necessarily be rendered easier of production in the upright posture, seeing that the direct influence of gravitation will aid the current; while, on the other hand, regurgitant murmurs at these orifices will be strengthened when, by lying down, the force of gravitation, which must act in opposition to the regurgitant current, is removed.

*Conduction of Sounds and Murmurs.*—The normal heart sounds are propagated to an extremely variable distance through the chest. We are by no means thoroughly acquainted with the physical facts which regulate the extent to which the sounds are conducted, but we know that if there be any alteration in the texture of the lung surrounding the heart, such a change invariably modifies the distance to which the sounds are carried. In cases of emphysema, for instance, the heart sounds are not conducted so far as in health, while in cases of consolidation they may be propagated to a considerably greater distance. We know, further, that in those individuals who have thin-walled chests, the heart sounds are conducted to a greater distance than in those whose thoracic parietes are invaded by fatty deposits.

The direction of propagation and the extent of conduction constitute a subject of much difficulty in the study of murmurs. It is quite true that there is one guiding principle—that of the better conveyance of sound in the direction of a moving current. This is, however, better exemplified in the case of the blood vessels than of the heart. As regards the latter organ, there are so many disturbing factors that it is hard to find any definite explanations of many results of auscultation. In the remarks which follow, some observations recently made by me will be employed to ascertain if it is possible to attain any adequate explanation of the conduction of murmurs.

The following illustrations have all been produced in the same way. After determining the area of cardiac dulness, the murmurs were mapped out on the surface of the body, and traced upon transparent paper applied to the chest. The figures were then reduced by photography to their present propor-

tions—one-seventh of the absolute size. The rhythm of the murmurs is shown by the shading :—vertical lines representing presystolic ; horizontal, systolic ; and diagonal, diastolic murmurs. The entire area over which such murmurs can be heard is shown by the extent of the shading. The point of maximum intensity is indicated by the circle containing stronger lines of the respective kind.

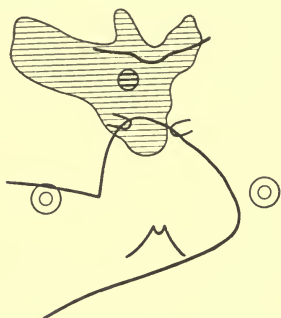


FIG. 58.—Aortic systolic murmur.

Dealing in the first place with systolic murmurs generated at the aortic orifice, it may be stated as a general rule that their point of maximum intensity is situated over the manubrium sterni at a considerable distance above what is commonly known as the aortic cartilage ; they are also more likely to be propagated along the clavicles and up the carotid arteries than in other directions.

Fig. 58 gives a good example of a simple aortic systolic murmur whose area of audition was mainly confined to this region.

The aortic diastolic murmur has a greater tendency to be propagated downwards, and its maximum intensity is very

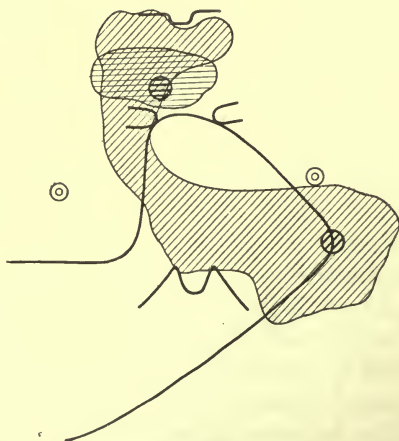


FIG. 59.—Aortic diastolic murmur.

commonly about half-way down the sternum, sometimes nearer the right, sometimes nearer the left border. But it

sometimes happens that the diastolic murmur may have its point of maximum intensity at a much lower level, and it may even be, as is shown in Fig. 59, situated at the apex of the heart. As is well known, a diastolic aortic murmur heard with its greatest intensity at the apex of the heart, has been held by Foster to indicate an affection of the left posterior cusp of the valve.

The total area of audition of the systolic murmur is sometimes co-extensive with the chest itself. The area of audition of the diastolic murmur occasionally presents curious outlines, as in the case from which Fig. 59 was taken. Some-

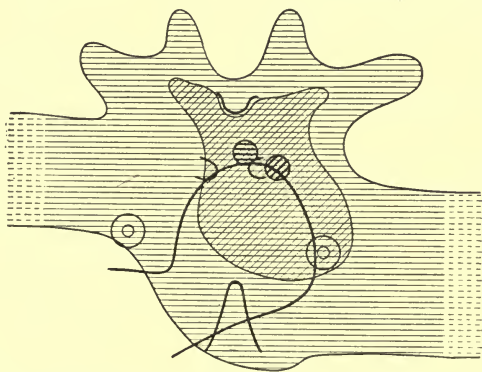


FIG. 60.—Aortic systolic and diastolic murmurs.

times aortic murmurs, both systolic and diastolic, have their greatest intensity to the left of the middle line. This occurrence, for which it appears impossible to offer any adequate explanation, will be more fully referred to in dealing with the diagnosis of aortic disease. Fig. 60 furnishes a good example from a case in point.

The different murmurs produced at the mitral orifice have their maximum intensity close to the apex beat. This is a rule to which there are very few exceptions indeed. The direction in, and the distance to, which these murmurs are propagated, are subjects of greater variation.

It is commonly believed that the presystolic mitral murmur is only propagated to a very short distance from the apex beat.

This, however, is not a statement to be accepted without reserve. In some instances, as in that shown in Fig. 62 and Fig. 63, the presystolic murmur is carried over a wide area, reaching

much beyond mid-sternum, in fact, as far as the right border of the heart. In the cases from which these figures were obtained there was nothing to complicate their study, as the

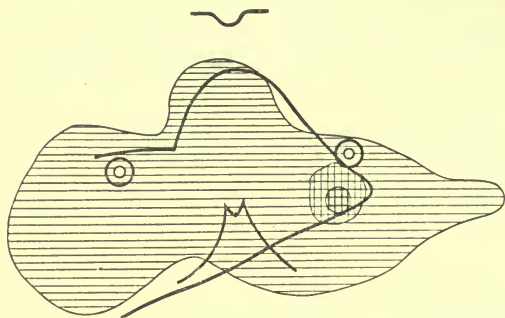


FIG. 61.—Systolic propagated more widely than presystolic murmur.

presystolic and systolic murmurs, both of mitral origin, were alone to be heard. In these cases the presystolic murmur had a wider area of audition than the systolic; in Fig. 61 and Fig. 64 the opposite and more common state of matters prevailed.

In cases presenting a diastolic as well as a presystolic murmur, the former murmur has sometimes a wider area of audition than the latter, at other times the converse is true. Fig. 65 is a good illustration of the former, Fig. 66 of the latter condition. In Fig. 65, in addition to the presystolic and diastolic mitral murmurs, there was found a blowing systolic murmur over the region of the conus arteriosus or pulmonary orifice. Fig. 66 was obtained from a young patient presenting solely presystolic and diastolic murmurs, and in whose case the diagnosis was that of pure mitral obstruction.

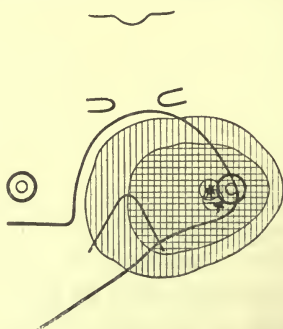


FIG. 62.—Presystolic propagated more widely than systolic murmur.

In Fig. 67 the presystolic and diastolic murmurs had an almost equal area of audition. There was in addition, however,



a systolic murmur heard over more than the entire præcordia, and having two points of maximum intensity, one near the apex, the other in the tricuspid area, at which points the characters of the murmurs were entirely different. The characters merged, however, into a somewhat uniform sound at a short distance from each point of maximum intensity.

Systolic mitral murmurs, like those of diastolic and presystolic rhythm, have their

maximum intensity at, or close to, the apex beat. They are conducted in every direction, but more commonly towards the axilla and the angle of the scapula. When combined

with other systolic murmurs they are usually all but blended into one another. But sometimes, as in Fig. 68, in which the mitral systolic was associated with an aortic systolic murmur, they remain discrete. On the other hand, as in Fig. 70, the systolic murmur, heard over almost the entire præcordia, and presenting four points of maximum intensity corresponding to the four areas,

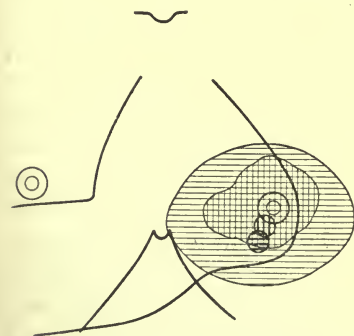


FIG. 64.—Systolic propagated more widely than presystolic murmur.

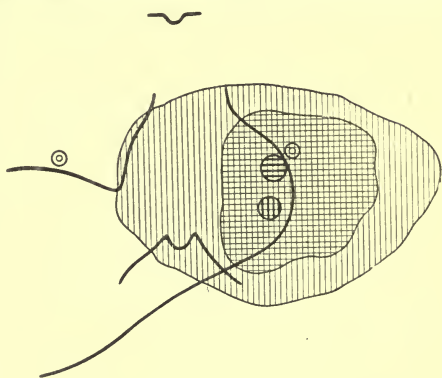


FIG. 68.—Presystolic propagated more widely than systolic murmur.

cannot be resolved into its constituent elements except close to each point of greatest intensity.

Pulmonary systolic and diastolic murmurs of organic origin have almost invariably their point of maximum intensity at the sternal end of the second left intercostal space. Those which

we shall afterwards see to be in all probability produced at the tricuspid orifice from relaxation of the cardiac muscle, may be situated somewhat lower down than is here mentioned, in fact,

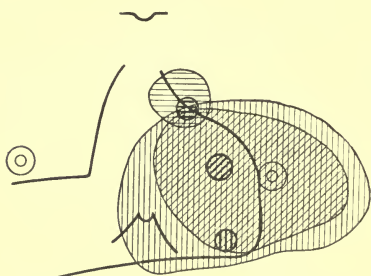


FIG. 65.—Presystolic propagated more widely than diastolic murmur.



FIG. 66.—Diastolic propagated more widely than presystolic murmur.

at any point between the recognised tricuspid area and that recognised as pulmonary. Such a murmur is shown in Fig. 71.

An excellent example of combined systolic and diastolic

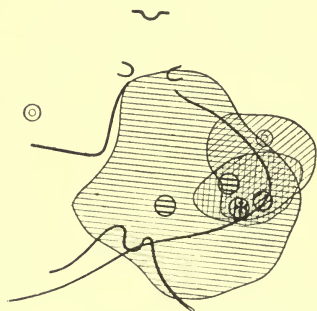


FIG. 67.—Presystolic and diastolic mitral murmurs with systolic mitral and tricuspid.

pulmonary murmurs of organic origin, full details of which will be given subsequently, is shown in Fig. 69. The diastolic pulmonary murmur produced, as will be more fully described in a later chapter, by dilatation of the orifice from strain without organic affection of the cusps, is not infrequently met with. Its position and extent may be made out in Figs. 72 and 73, in the former of which it

was associated with presystolic and systolic mitral murmurs, while in the latter it was accompanied by systolic murmurs in the mitral, tricuspid, and pulmonary areas.

Tricuspid murmurs are usually heard most distinctly at the junction of the lower left costal cartilages with the sternum, that is over the superficial cardiac dulness. In an excellent

example in which tricuspid and mitral lesions co-existed, the distribution of the murmurs was as is shown in Fig. 74; in another instance, in which there was a loud pre-systolic murmur heard over a wide area with its maximum intensity close to the edge of the sternum at the level of the fourth costal cartilage, it was impossible to come to any other conclusion than that there was obstruction of both orifices. The full details of this case are given below. It is shown in Fig. 75.

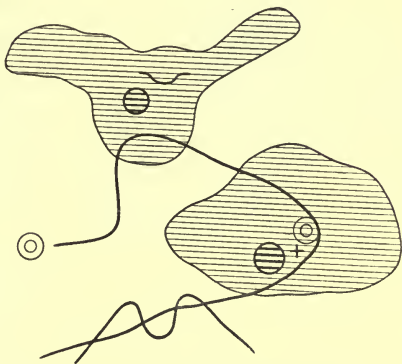


FIG. 68.—Systolic aortic and mitral murmurs.

The systolic tricuspid murmur has its maximum intensity about the fifth left chondro-sternal articulation, and is conducted from that point in every direction. It is, however, very common to find that it is loudest somewhat higher up; the exact significance of this will be discussed later.

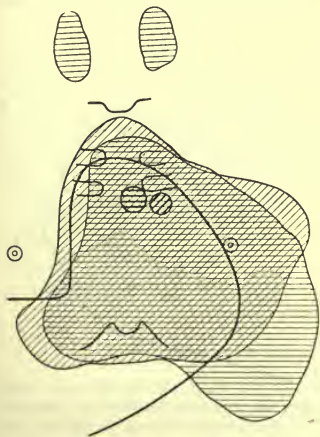


FIG. 69.—Systolic and diastolic aortic and pulmonary murmurs.

There can be no doubt that the conduction of murmurs is chiefly dependent upon the vibrations produced in the chest wall. It is not easy, however, to decide what particular kind of murmur is likely to be best carried in this way.

Certainly it is not always the murmur of loudest intensity. In several instances this was tested in the cases referred to. For instance, the case furnishing Fig. 66 presented greater

intensity of the presystolic than of the diastolic murmur, as tested by the method of Vierordt, that is, by the interposition

of non-conducting plates between the chest of the patient and the ear of the observer. Notwithstanding this greater intensity of the presystolic murmur, it was not conducted so widely as the diastolic murmur. Exactly the converse was found in the case shown in Fig. 65; here the diastolic murmur was of louder sound but more restricted distribution. That the presystolic is sometimes, if not indeed

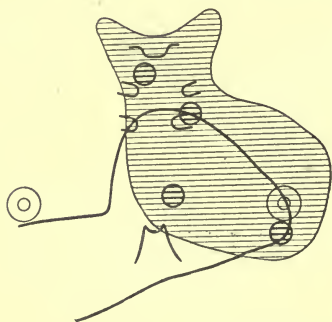


FIG. 70.—Systolic murmur with maximum intensity in four areas.

often, carried more widely than the systolic murmur can be seen in Figs. 62, 63, and 74.

It may be accepted as certain that the mode of conduction of any murmur is in the first place conditioned by the cardiac tissues in the immediate neighbourhood of the orifice at which it is produced. Some of the valves, the aortic for instance, have intimate relations with the interventricular septum, by means of which vibrations, communicated from the blood, can be transmitted to the whole heart. Other cusps, as the pulmonary, are not so closely associated with the muscular substance of the heart, and sound vibrations must be less easily communicated to it. Some of the recent observations of Ewart and Habershon on the conduction of the sounds of the heart may doubtless be usefully extended to the difficult problem of murmur propagation.

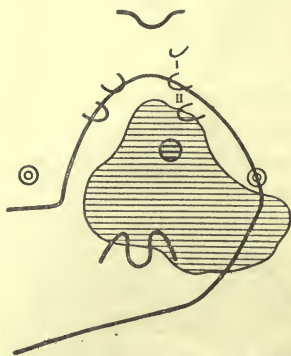


FIG. 71.—Systolic murmur of tricuspid origin heard most distinctly near pulmonary area.



In the remarks which have been made on this subject the usual method of studying the sounds and murmurs, as

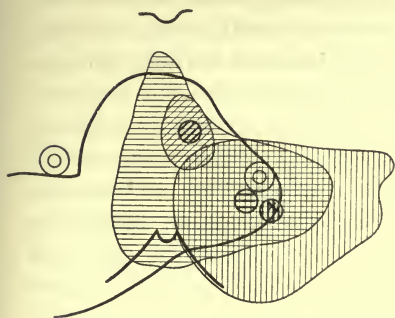


FIG. 72.—Presystolic and systolic mitral with diastolic pulmonary murmur.

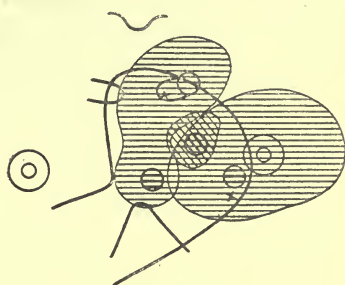


FIG. 73.—Systolic mitral and tricuspid murmurs with diastolic pulmonary murmur.

practised in this country, has been followed. No advantage can, in my opinion, accrue from the adoption of the method of

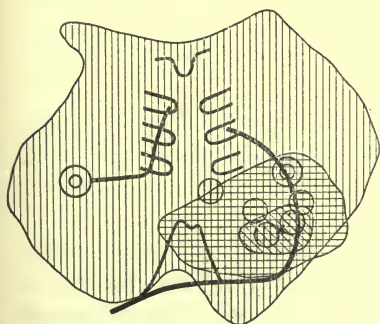


FIG. 74.—Presystolic systolic and diastolic mitral and presystolic and diastolic tricuspid murmurs.

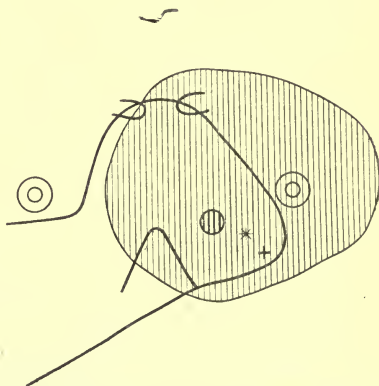


FIG. 75.—Tricuspid and mitral presystolic murmur.

Potain, according to which the præcordia is divided into apical, mesocardiac, and basic regions. These three main regions are respectively subdivided into (1) apical proper, parapical, endapical, and supra-apical; (2) left ventricular, xiphoid, and sternal; and (3) pre-aortic, and pre-

infundibular. Such a system is faulty in many ways, but chiefly by reason of confusing external landmarks and internal structures in its nomenclature. It is surely much better to follow the established terminology, such as is well given by Ewart, than to lend any countenance to a scheme of doubtful utility.

This is probably the most fitting place in which reference may be made to the method of investigation termed auscultatory percussion, which has been recently employed and recommended. Having by repeated observations convinced myself of the absolute accuracy of our ordinary mode of percussion, as regards the heart, the plan of auscultatory percussion does not commend itself to my judgment. It is satisfactory to find myself in entire agreement with the opinion of Broadbent on this point, as the method is of absolutely no utility or value in any respect.

#### SYMPTOMS CONNECTED WITH THE VESSELS.

The appearances connected with the peripheral circulation furnish symptoms of great importance; in fact, they yield evidence of the highest diagnostic value. The most systematic course in considering the subject is to deal separately with the arteries, capillaries, and veins.

THE ARTERIES.—The physiological factors concerned in the production of the arterial pulse have been sufficiently detailed, and the clinical phenomena to which they give rise remain for consideration in this place.

*The Pulse.*—The arterial pulse has received the attention of the physician from the early days of medicine, but it is impossible that there can have been any real appreciation of the indications which it furnishes until the discovery of the circulation of the blood.

Hippocrates, according to Adams, makes no mention of the pulse, but in the edition of Littré several references to its different conditions may be found. Aristotle observed that the pulse throughout the whole body was simultaneous; that the heart beat and arterial pulse were synchronous became known to Herophilus. The last-mentioned author described the different qualities of the pulse under the headings of size, frequency, force, and rhythm. Rufus in a short treatise analysed many of the properties of the pulse, and recognised such characters

as size, rapidity, frequency, strength, and resistance, besides naming certain special types of pulse. It is of much interest to note that he recognised the movements of the fontanelles in children to be produced by arterial pulsation. Galen spent a considerable amount of his indefatigable industry upon the pulse, and produced seven distinct treatises on the subject. As regards the circulation, his most important work was the discovery that the arteries contained blood. He also, however, observed the influence of many factors on the pulse. With an excessive degree of subtlety he analysed its different features. Some of the most important of his opinions are summarised by Broadbent in his work on the pulse, in which he justly remarks of Galen that the general effect of his writings "is to confuse the essential features of the important variations of the pulse by overwhelming them in minute distinctions of no practical significance. Indeed, his point of departure is not observation, but theory, and the varieties are not described from nature, but deduced from axioms."

No progress was made in regard to the study of the pulse until the appearance of Harvey's immortal work rendered the comprehension of its causation possible. Notwithstanding the light thrown by his investigations upon the relation of the heart beat and the arterial pulse, no real advance in the investigation of the latter was made until the time of Hales, who for the first time attempted to estimate the arterial pressure by means of a glass tube introduced into the aorta of the horse. He was followed in these investigations by Vierordt and Poiseuille, but it is more particularly to Ludwig and Fick that we are indebted for methods of investigation of the arterial pressure.

Wilkinson King in the early years of the present century demonstrated variations in the contents of the veins by means of slender rods of glass or shellac, and Vierordt, following in his footsteps, placed a straw across the arm, by means of which he traced the movements of the radial artery upon a travelling surface. Such were the beginnings of the sphygmograph evolved by Vierordt from such simple methods. It is principally to Marey that we are under obligations for the development of sphygmography, but in addition to him must be mentioned Sanderson, Foster, Mahomed, Landois, Sommer-

brodt, Roy, and von Frey. The development of our knowledge connected with this branch of the subject, as well as with the characters and factors of the arterial pulse, may be studied in the works of Landois, Ozanam, Broadbent, and von Frey.

*Inspection of the Pulse.*—The arterial pulse may be observed by means of inspection. In healthy youth the pulsation of the arteries can only be seen in a few situations. It usually is seen in the carotid arteries of the neck; it is frequently visible in the temporal artery; it may sometimes be detected in the radial artery above the wrist. In elderly people, more especially when thin, pulsation in many of the arteries may be seen, and in conditions producing considerable changes in the structure of the arterial walls, or in the variations of arterial pressure, inspection affords useful indications. In arterio-sclerosis, for example, the temporal arteries may be seen pursuing a tortuous course upon the sides of the face and head. Many of the superficial arteries of the limbs may also present the same feature, while in aortic incompetence the great variations of arterial pressure make themselves manifest by the excessive pulsation of all the superficial arteries, which can, however, be best seen in the arteries of the neck.

*Palpation of the Pulse.*—Of much greater importance from the point of view of practical medicine is the observation of the arterial pulse by the sense of touch. The arterial pulse may be investigated with the finger most satisfactorily in the radial artery. The part of the vessel most available for the purpose of observation is that which lies immediately above the wrist, between the prominent ridge of the radius on the outer, and the flexor tendons of the hand on the inner side. The arteries are in this situation only covered by the skin and the subcutaneous tissues, and they are therefore particularly well adapted for investigation. As the artery, moreover, rests almost immediately upon the radius at this point, there is a firm base against which it may be pressed, and in this way many of its features may be brought out with greater ease.

The condition of the pulse should always be ascertained while the patient is sitting or reclining, and the arm which is employed for the purpose of determining the condition of the pulse must be allowed to rest upon some object, or must be



supported by the hand of the observer, which is not made use of in palpating the artery. The observer should place himself either in front, or to the right, of the patient, whose right arm ought to be in the semi-prone position with the elbow slightly flexed; he must then pass his right hand across the radial surface of the patient's wrist and lay the tips of his index, middle, and ring fingers upon the radial artery. If the patient's left radial artery is to be examined the observer should stand to the left and employ his left hand in a similar manner; if both radial arteries are to be examined at the same time he should, as far as possible, stand in front of the patient and use his right hand for the right artery and his left for the left. Experience teaches that the pulse may be best palpated with three fingers in such a way that the index finger will be nearest to the heart, and thus be able to produce changes in the pulsation that may be appreciated by the other fingers. Attention to these details may seem unnecessary, but the adoption of such a method will be found to render the examination of the pulse at once more easy and more precise.

*The Sphygmograph.*—The invention of the sphygmograph, by means of which the graphic method of investigating movements has been applied to the arterial pulse, was at one time regarded as affording high promise of clinical utility. These anticipations have unfortunately not been fulfilled. It is perfectly true that the sphygmograph has been of great value in throwing light upon some points previously obscure in the physiology of the circulation, and in clinical research it must be regarded as being of some service as a means of demonstrating certain qualities of the pulse. It is, nevertheless, unnecessary for diagnosis, and useless in prognosis; it is therefore of no value in the treatment of disease. It has revealed no new fact by means of which affections may be discovered that were unknown before its invention. It brings into prominence, however, certain points which are less definite without its aid, and it is therefore on this account of some clinical interest. It must be remembered that there are some aspects of the pulse which can be much more accurately estimated by means of the finger than by the help of the sphygmograph, and the most enthusiastic advocate of this

instrument cannot regard it as being more than a supplement to the finger of the observer. It is to be borne in mind that tracings taken by the sphygmograph from the same artery and at the same time, but by means of different forms of instrument, or even by different persons employing the same instrument, may present absolutely different appearances.

The best forms of sphygmograph are all in their essence based upon the model adopted by Marey, which is still deservedly a favourite with those who make use of the instrument. The chief differences as regards the mechanism of the sphygmograph lie in the method by which the pressure of the instrument is adapted to the vessel. In Marey's original instrument, and in its modifications by Mahomed and von Frey, the pressure is regulated by means of a spring; while in the sphygmograph devised by Sommerbrodt the pressure is managed by means of weights bearing directly upon the pad which rests on the artery. Several other forms of sphygmograph are in use, but none of them are so reliable as those which have been referred to. It is therefore unnecessary to deal with them. In the application of the sphygmograph care must be taken to have the patient in a position of ease. If not lying in bed, or reclining on a couch, he ought to be seated comfortably. The arm and hand must rest easily upon the pad employed for the purpose, with the arm supinated, the wrist bent slightly backwards, and the fingers semi-flexed. This position brings the artery into greater prominence and relieves it from interference by the adjacent tendons. A line is then to be drawn with the clinical pencil along the course of the radial artery, so that it is easy to ascertain at any moment if the instrument is in proper position. After winding up the clockwork of the instrument, it is to be applied to the wrist in such a way that the ivory pad lies over the radial artery, on the inner side of the styloid process of the radius. The paper, blackened by smoke from a piece of burning camphor, or from a lighted candle, is to be placed in its proper position by means of the frame. The point of the pen must then be adjusted so that in its oscillations it will occupy the central part of the paper. By means of the spring or weights, according to the

type of instrument employed, the pressure is to be adjusted so as to bring out the largest amplitude of movement, and the tracing is then to be taken by starting the clockwork. Some of the features ascertained by means of the sphygmograph will be referred to in detailing the characters of the pulse in the succeeding pages.

To show how tracings obtained from the same artery at

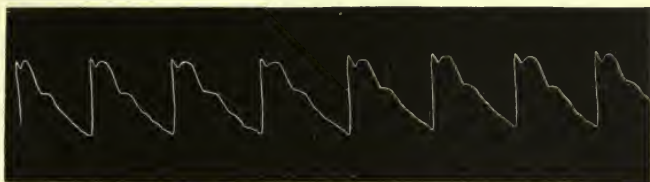


FIG. 76.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral incompetence ; pressure 2 oz.

the same time and by the same observer, but with different instruments, may differ, the accompanying curves (Figs. 76 and 77) are useful. They were obtained by me from a patient suffering from mitral incompetence.

The normal characters of the radial pulse vary of necessity

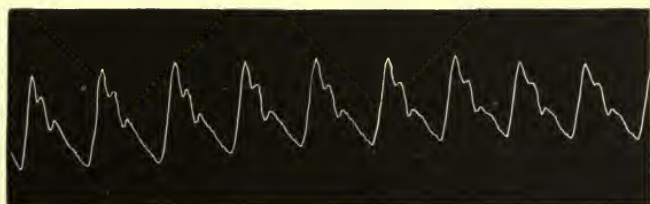


FIG. 77.—Tracing taken with Sommerbrodt's sphygmograph from the radial artery in a case of mitral incompetence ; pressure 2 oz.

within wide limits, but it may be said that the wall of the artery should be yielding, yet elastic, while the vessel should be well filled with blood, although easily compressible. The rate of pulsation exhibits great differences in frequency according to circumstances, but the rhythm of the pulsation should be absolutely regular. Each individual pulsation should be of moderate and uniform size and force, while neither too long nor too short in duration.

A sphygmographic tracing of the pulse in health, as shown in Fig. 78, is characterised by a sudden and uninterrupted line of ascent and a much more gradual line of descent marked by two distinct undulations. These points are diagrammatically shown in Fig. 79. The line of ascent, *a-b*, inclines slightly



FIG. 78.—Tracing from pulse of healthy man aged 36; pressure  $2\frac{1}{2}$  oz.

forwards when obtained by means of sphygmographs writing, like Sommerbrodt's, with a lever at right angles to the paper. It may be perfectly vertical or even inclined somewhat backwards when obtained with sphygmographs, which, like Marey's, write with the lever in the axis of the travelling paper. This

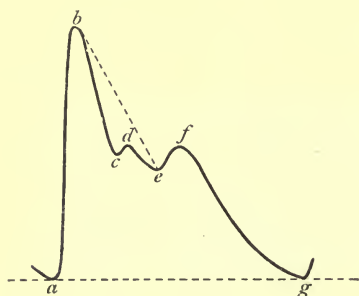


FIG. 79.—Diagram of pulse tracing in health.

line of ascent is commonly called the percussion wave, and it results from the sudden entrance of the blood from the left ventricle into the arterial system. It is in no sense to be regarded as caused by a wave of blood. It is entirely produced by the wave of increased pressure resulting from the cardiac energy

which drives the blood into the aorta.

The height of the up-stroke or percussion wave depends on the extent of pressure produced in the arteries by the influx of blood from the left ventricle, and it must be conditioned by the volume of blood issuing from the ventricle, by the amount of outflow into the capillaries, and by the resistance of the arterial walls. The termination of the up-stroke would naturally be expected to pass gradually into the line of descent, and the fact that it as a rule ends in a sharp apex has usually been



accounted for by the inertia of the sphygmograph. Roy and Adami, however, believe that the sharp apex is produced by the contraction of the papillary muscles, and adduce in support of this view comparative tracings from the root of the aorta and the left ventricle of the dog. As has been previously shown, their views on the contraction of the papillary muscles cannot yet be accepted, at least until further evidence in support of them is advanced.

The line of descent (*b-g* in Fig. 79) results from the gradual diminution of arterial pressure after the cessation of the cardiac systole. It is interrupted by two important elevations. The first of these (*c-d*) is often termed the tidal wave, or, avoiding theory, the predicrotic wave. It is commonly believed to be produced by the current of blood passing along the artery, and according to Roy and Adami this portion of the pulse curve agrees in form with the intra-ventricular curve. It is termed by them the outflow-remainder wave. To this succeeds a second interruption of the line of descent (*e-f*), which is universally accepted as due to an onward wave of increased pressure resulting from the recoil of blood upon the aortic cusps. This recoil is consequent upon the diminution of pressure in the first part of the aorta, after the cessation of ventricular systole, which is produced by the inertia of the column of blood, as is beautifully described by Foster. Both of these secondary waves are found to be higher upon the line of descent in the case of arteries close to the heart than in the case of those situated at a greater distance. The sphygmographic curve simply gives an approximate representation of the variations of pressure within the artery, and furnishes no measure of the fulness of the vessel. The sphygmograph, from what has been said, can only be regarded as a means of amplifying the information obtained by the finger in regard to one or two aspects of the pulse.

*Method of Studying the Pulse.*—To facilitate the investigation of the arterial pulse the various details should be considered in a definite order, and the system which is adopted in the following pages is that which experience has taught me to regard as giving the most satisfactory results.

*The Wall of the Vessel.*—The condition of the arterial

walls should in every case be first investigated. In health the vessel should be yielding, giving at the same time a feeling of elasticity to the fingers. Any departure from this state of matters must be regarded as abnormal. In febrile conditions and in many states of malnutrition the walls of the artery are too yielding, and do not furnish the healthy feeling of elastic return on diminishing the pressure of the finger. On the other hand, the artery may be so hard and resistant as to roll from side to side under the pressure of the finger. This condition is common in advanced years, and depends upon arterio-sclerosis, often associated with chronic renal disease, and attended by cardiac changes.

It is always advisable to pass the fingers up the forearm in order to ascertain if there is any sinuosity or tortuosity of



FIG. 80.—Tracing taken from a case of advanced atheroma of the arteries ; pressure 3 oz.

the artery. Such conditions are very common in arterio-sclerosis, and are produced by lengthening of the vessel through changes involving growth of fibrous tissue, by means of which loss of distensibility and elasticity is compensated by increased rigidity. Thoma holds that the growth of the connective tissue produces an increase of elasticity. In this use of the term, however, he is clearly in error, as has been elsewhere pointed out by myself.

It is not always easy to distinguish this condition from high arterial pressure, but a state of high pressure without any change in the vessel-wall is unaccompanied by any hardness of the artery, and there is no sinuosity or tortuosity in it.

Sphygmographic tracings obtained from patients exhibiting the features of arterio-sclerosis show a moderate up-stroke, with a blunted apex, and little tendency to any interruptions of the curve during the line of descent, as may be seen in the illustration (Fig. 80).

The limited range of movement, the blunt appearance of the summit, and the absence of the secondary waves on the down-stroke are produced by the rigidity of the arterial wall.

*Blood Supply.*—The state of the blood supply may be estimated by ascertaining the fulness of the artery, and the degree of blood pressure. These two features of the pulse might be expected to stand to each other in some definite relation; this, however, is not the case. Both depend upon the three great factors already mentioned—the amount of blood in the arteries, the degree of cardiac energy, and the resistance in the arterioles; differences, nevertheless, in the relations borne by each of these factors to the others may produce such apparently paradoxical conditions as a full pulse of low pressure, or an empty pulse of high pressure. The former may sometimes be seen in chronic renal disease, the latter is occasionally found in peritonitis.

The fulness of the vessel is to be judged by its size during the interval between two pulsations, and the educated touch is the only means by which this may be determined, the sphygmograph rendering no assistance in this respect. A full artery (*pulsus plenus*) may be attended by large pulsations, in which case there is of necessity only moderate or even low arterial pressure; it is, however, more common to find with full arteries that the pulsation is small. A full pulse is found in those who present increased resistance in the arterioles and capillaries, as may be seen in many conditions found in those commonly termed plethoric. It is also observed in the early stages of arterio-sclerosis and chronic renal disease. An empty artery (*pulsus vacuus*) is often found associated with a large and bounding pulse, as in that characteristic of aortic incompetence. An empty pulse is also found in many conditions of relaxation of the small arteries and arterioles. In convalescence from acute affections, in wasting diseases, and in malnutrition the pulse is almost invariably empty.

The blood pressure is to be estimated by the amount of force necessary to obliterate the artery during the interval between the pulsations. A pulse of low pressure is compressible, while one of high pressure is incompressible. To some extent this quality may also be estimated by the amount

of expansion which the artery undergoes during the cardiac systole, a pulse of high pressure being less distensible than one of low pressure. Sphygmographic tracings furnish important indications in regard to pressure, seeing that the size of the tidal, or outflow-remainder, wave stands in direct relation to the extent of pressure. If a line be drawn from the summit of the tracing to the lowest point of the dicrotic notch, the tidal wave sometimes does not reach it, and at other times it passes beyond it. If it be below it, as in Fig. 81, the pulse is of low or moderate pressure; but if, as in Fig. 82, it is higher than the line, the pulse is of high pressure. With high pressure the pulse wave is usually small, but this is by

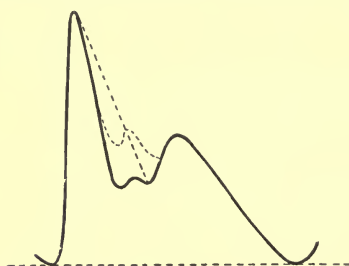


FIG. 81.—Diagram of pulse of low pressure.

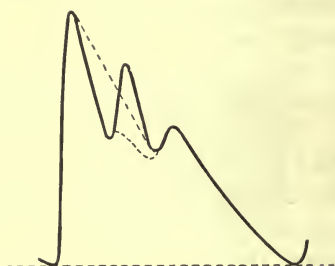


FIG. 82.—Diagram of pulse of high pressure.

The finely-dotted line represents the normal curve.

no means invariably the case, and although with low pressure the pulse wave is very commonly large, yet in many cases it remains small.

High pressure is found in many circumstances which produce increased resistance in the arterioles, such as the early stages of acute diseases, such diathetic conditions as lithæmia and arterio-sclerosis. Low pressure is, on the other hand, found in the later stages of acute diseases, and in many conditions of malnutrition; it is also found in conditions which impair cardiac energy, such as aortic and mitral lesions—particularly aortic incompetence and mitral obstruction.

It might be thought that arterio-sclerosis would be incompatible with low pressure. This, however, is not the case, and although the proof is not easy in advanced changes it is not difficult in earlier stages. The annexed tracing (Fig. 83) is



from a patient with a considerable degree of arterio-sclerosis; it nevertheless shows the characteristic features of low pressure.

One of the most prominent features resulting from low pressure is dicrotism. On palpation of the radial artery under

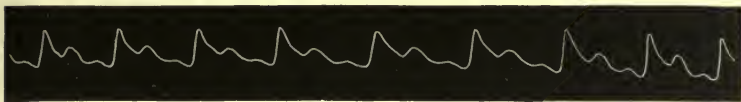


FIG. 83.—Tracing from radial artery showing low pressure with sclerosis of vessels; pressure 2 oz.

ordinary conditions only one pulsation can be felt corresponding to each cardiac systole; in certain conditions, however, a second wave can be felt immediately following it. A tracing of such a pulse is shown in Fig. 84. It reveals an entire

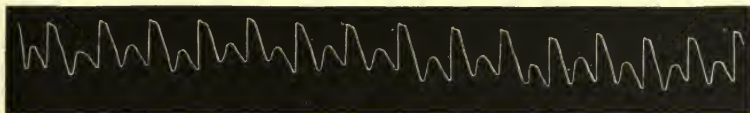


FIG. 84.—Pulse from a case of pericarditis showing dicrotism; pressure  $1\frac{1}{2}$  oz.

absence of the tidal wave with an exaggeration of the dicrotic notch. When the notch reaches the base line of the tracing, as is shown in the diagram (Fig. 86), the pulse is said to be fully dicrotic. When the notch sinks below the

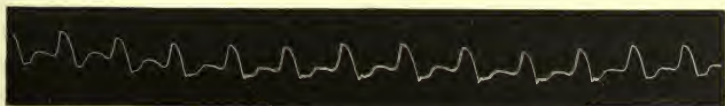


FIG. 85.—Pulse from a case of enteric fever showing hyperdicrotism; pressure 2 oz.

base line, as in Fig. 85 and Fig. 87, the pulse is said to be hyperdicrotic.

That the condition of dicrotism is the direct result of low pressure has been frequently proved by such observations as those of Winternitz on the effect of hot baths on the pulse; the result being to change a pulse manifesting all the features of high pressure, with a well-marked tidal wave, into one of low pressure, with extreme dicrotism. The condition is more

particularly found as a clinical feature in the later stages of acute febrile affections.

*Nature of Pulsation.*—The character of the pulsation has to be considered in regard to the rate and the rhythm of the pulse.

The rate of the pulse is to be estimated by the number of pulsations in a given interval of time, as was first introduced, it is interesting to remember, by Kepler, the great astronomer. It is subject to alterations in the cardiac energy, the blood supply, and the arterial tone. In an adult man the number of pulsations is usually between 60 and 70 per minute, but

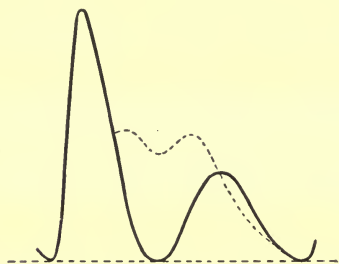


FIG. 86.—Diagram of fully dicrotic pulse.

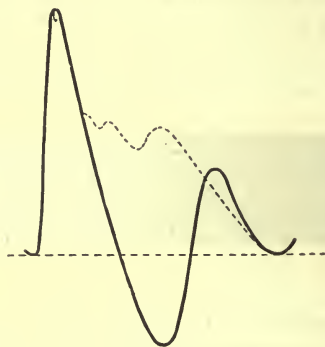


FIG. 87.—Diagram of hyperdicrotic pulse.

The finely-dotted line represents the normal curve.

there are conditions of apparent health in which the pulsations may sink to 20, or rise to 100 per minute. An increase in the rate constitutes a frequent pulse (*pulsus frequens*), while the diminution is termed an infrequent pulse (*pulsus rarus*). The terminology commonly employed as regards the rate of the pulse is unfortunately very lax, the terms frequent and quick being used indiscriminately, as if they denoted the same quality. The real meaning of quick will be mentioned below, and all that need be said here is that the true phrase for a pulse which occurs often in a given interval of time is frequent—not quick.

Sphygmographic tracings give a permanent record of the frequency of the pulse, but it must never be forgotten that the number of pulsations shown by any tracing not only depends

upon the rate of the heart but on the speed of the clockwork. It is only when the speed is uniform that two tracings can be compared with any degree of usefulness. Fig. 88 shows an extremely frequent pulse in aortic incompetence along with acute pneumonia. It also brings out some irregularity in rhythm, as well as the collapsing character of each pulsation.

The rate of the pulse in health varies in accordance with many circumstances, more particularly in regard to the sex, age, size, and position of the individual, the external temperature, the hour of day, the condition of digestion, the occupation of the moment, and other less important factors. It may be said that as a rule the pulse is more frequent in the following conditions:—In the female sex; in infancy and childhood; in small persons; in the upright attitude; in high temperatures;

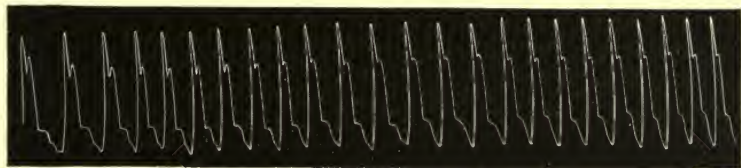


FIG. 88.—From acute pneumonia in a patient suffering from aortic regurgitation ; pressure 2 oz.

during the later periods of the day; after eating and drinking; and during mental or physical exertion.

The rate of the pulse is increased in pyrexia, and the increase is in most diseases directly proportional to the rise of temperature; in cerebral inflammation and in enteric fever this direct ratio is frequently absent. The pulse is also increased in frequency in certain nervous diseases, especially in exophthalmic goitre and in nervous palpitation. It is also more frequent in almost all the organic diseases of the heart, the only common exception being obstruction of the aortic orifice.

The rate of the pulse undergoes a diminution in frequency during, and following, the later stages of some fevers, more particularly if there is any tendency to collapse; in certain toxic states of the blood, such as jaundice; in most conditions of high pressure, such as occur in renal disease; in some affections of the muscular structure of the heart; and in certain affections of the meninges.

It is probable that in conditions of absolute health the pulse is perfectly rhythmic, that is to say, it occurs with equal intervals between its beats, but it is certainly true that the pulse even in absolute health may be very irregular. The rhythm depends primarily upon the automatic contractions of the heart walls, which are, however, subject to interferences produced by the influence of the nervous system. It is prob-



FIG. 89.—Tracing from the radial artery in a case of mitral stenosis; pressure  $2\frac{1}{2}$  oz.

able that any interference with the access of blood to the heart will, by disturbing the normal stimulus, produce disturbances of the automatic contraction, as in mitral obstruction, from which the accompanying tracing, Fig. 89, was obtained. It is also certain that nervous influence frequently gives rise to irregularity. Irregularity of the pulse has at times so much periodicity in changes as to give the appearance of a second

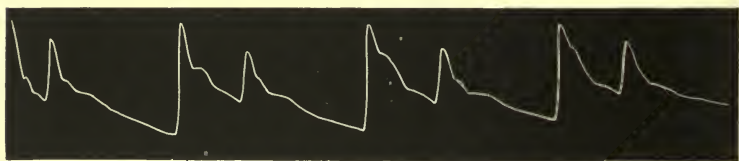


FIG. 90.—*Pulsus bigeminus et alternans* in aortic disease; pressure  $2\frac{1}{2}$  oz.

and larger rhythm superposed upon the original. As examples of what is meant by this remark, may be cited intermission, or the failure of some of the beats, giving rise to the *pulsus deficiens*; and intercurrence or the intercalation of additional pulsations, resulting in the *pulsus intercidens*. It sometimes happens that such a periodic tendency is even better seen, as when the pulse occurs in pairs of beats, when it is termed the *pulsus bigeminus*, or in threes, when it is known as the *pulsus trigeminus*. On the other hand, a large and a small pulsation sometimes occur time about, when the pulse is termed *pulsus*



*alternans*. A good example of the bigeminal pulse is shown in Fig. 90, obtained from a patient suffering from cardiac failure in aortic disease.

Simple irregularity of the pulse is often a result of nervous diseases, and is found quite commonly at both extremities of life. In young persons it may simply be the result of the adaptation of the different functions of the body, but in elderly people it is usually an expression of some alteration in the circulatory apparatus. In mitral obstruction, irregularity of the pulse, giving rise to the most marked arrhythmia, is a frequent occurrence. It does not, as a general rule, make its appearance until somewhat late in the development of the disease; but this rule has frequent exceptions, and the irregularity sometimes begins at an early stage of the disease. It is probable that its production is associated with an increased stimulus to the auricles on account of the pent-up blood. In degeneration of the walls of the heart many of the forms of irregularity which have been mentioned occur, and frequent opportunities have been afforded me of observing rhythmic variations in the size, as well as in the regularity, of the pulse in the fatty heart.

In many instances of irregularity some of the pulsations of the heart are not sufficiently powerful to give rise to pressure such as may be experienced in the peripheral arteries. On listening to the heart sounds with the finger on the radial artery, many of the heart sounds are found in such instances to be unfollowed by any beat of the radial pulse. Such cardiac pulsations are said in cases of this kind to be abortive, and they are a frequent cause of intermission of the pulse.

*Character of Pulse Wave.*—The character of each individual beat of the pulse yields much information in regard to the state of the circulation. The size of the beat produced by the wave of increased pressure depends of necessity upon the relations of the three factors concerned in the production of the pulse. Speaking generally, it may be said that the size of the wave produced by the increased pressure is larger in conditions attended by emptiness of the arteries, with increased energy of the heart. A large bounding pulse (*pulsus magnus*) is therefore found in conditions of arterial relaxation, as in

pyrexia, and instances of cardiac hypertrophy without constriction of the arterioles, as sometimes occurs in cases of Graves' disease. This form of pulse is seen in a very marked fashion in many cases of aortic incompetence, in which, as a result of the regurgitation, there is at once a tendency to emptiness of the arterial system, along with dilatation and

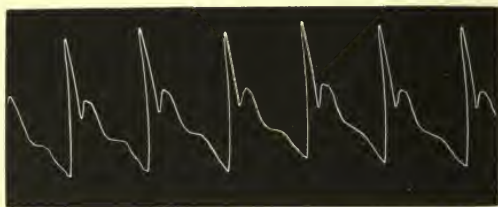


FIG. 91.—Tracing from the radial artery in a case of aortic incompetence ; pressure 3 oz.

hypertrophy of the left ventricle. It must be remembered however, that in this affection there may be some interference with the development of such a characteristic pulse on account of obstruction of the aortic orifice, while alterations in the resistance in the arterioles as well as in the energy of the heart may produce considerable alterations in it. A characteristic tracing of such a pulse is shown in Fig. 91.

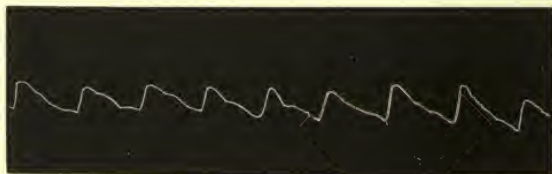


FIG. 92.—Tracing from the radial artery in a case of aortic stenosis ; pressure  $2\frac{1}{2}$  oz.

A small or thready pulse (*pulsus parvus*) occurs in inanition, cardiac weakness, and obstruction in the arteries or arterioles. It is sometimes very characteristic of aortic obstruction, as is shown in Fig. 92.

In health the volume of the pulse increases on inspiration and diminishes during expiration. The reverse is occasionally observed, *i.e.* diminution of the volume during inspiration and increase on expiration. This condition of the pulse is termed *pulsus paradoxus*, and was originally considered to be due to

mediastino-pericarditis. This, however, is by no means absolutely true, and Fig. 93 shows an excellent example of this form of pulse in simple adherent pericardium. The tracing is marked with *e* at each expiration, and *i* on inspiration.

The force of each increased pressure wave has no necessary relation to its size. The large and bounding pulse found in low pressure is often very feeble, while the small and wiry pulse of increased pressure is frequently possessed of considerable force. The amount of force depends mostly on the degree of tone possessed by the arterioles, but also to some extent upon the energy of the cardiac systole, and the result is conditioned by the relations of these two factors to each other, the amount of blood in the arteries having less effect.

The duration of each beat depends upon the relation

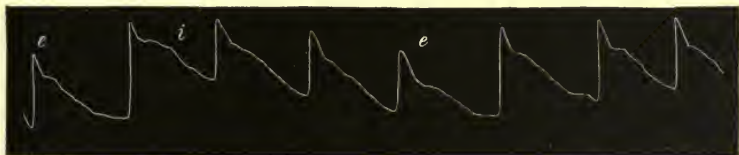


FIG. 93.—*Pulsus paradoxus* in adherent pericardium ; pressure 3 oz.

existing between the expansion of the artery by the wave of increased pressure and its contraction during the onward passage of the blood. Its duration is longer when the blood encounters considerable resistance in passing into the capillary circulation, and it is shorter under converse circumstances. It is necessarily longer when the systole of the heart is lengthened, and shorter when the cardiac contraction is brief. A pulse of long duration is correctly termed a slow pulse (*pulsus tardus*), while one of short duration is called quick (*pulsus celer*). As was mentioned before, these terms are often misapplied, the phrase quick being applied to a pulse which is frequent. As tested by the finger the quick is readily distinguishable from the slow pulse by the duration of the impact. The slow pulse gives a feeling of persistence, while the quick pulse gives rise to a sensation almost as evanescent as would be produced by the impact of a small body striking the finger and ricocheting off.

A slow or sluggish pulse is almost invariably associated with high arterial pressure; this, however, is not an absolute rule, as, in aortic obstruction for example, when there is much obstruction the pulse may be extremely sluggish, although the pressure is low. The slow pulse is found in all affections where there is contraction of the smaller arteries, as in arteriosclerosis and renal disease.

A quick or active pulse is found as a rule along with low arterial pressure, and it depends mostly upon a relaxed condition of the arterioles. It is therefore extremely common in febrile affections. A very marked example of the quick pulse is to be observed in cases of free aortic regurgitation, where the celerity of the pulse is associated with an empty condition of the artery and a large pulse wave. These conditions are much more distinctly marked when the arm is raised, and in all cases of suspected aortic regurgitation the physician should support the patient's arm at as high a level as possible with one hand, while he employs the other to investigate the pulse. The reason for this is so obvious as to require no further remark.

In conditions of health the individual pulsations are approximately equal in size, force, and duration; when there are irregularities in rhythm there are almost invariably inequalities in size and force.

*Pulse in different Vessels.*—The characters of the pulse in different arteries should always be investigated in order to determine whether there be any local interferences with the arterial circulation. In similar arteries on opposite sides of the body the pulse under ordinary circumstances occurs at the same instant of time, and if any difference is present some obstacle must be the cause of the delay in the pulse which occurs later. In the same way there is a definite interval of time between the pulse in arteries situated at different distances from the heart, between the carotid and radial, for instance, or between the radial and femoral or tibial. This was apparently first observed by Weitbrecht and has been the subject of numerous observations since the date of his researches, Weber, Czermak, Landois, Grunmach, Keyt, Edgren, and Hoorweg having devoted much attention to the subject. As the general



result of these investigations it may be said that the period of time elapsing between the carotid and radial pulse is about  $\cdot 08$  sec., while between the carotid artery and the dorsal artery of the foot it is about  $\cdot 13$  sec.

The relation in time between the pulse in different vessels must necessarily be learned by practice. If it should be altered, there must be some cause of interference leading to the

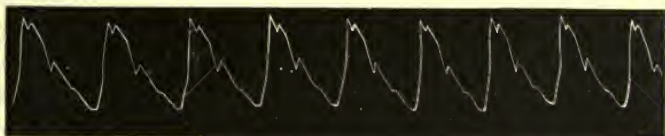


FIG. 94.—Tracing from right radial artery.

change. When there is any delay in the pulse of any artery there is also at the same time some alteration in its characters. In cases of thoracic tumours or aneurysms, for example, there may be a considerable difference in time and volume between the pulse of the right and left radial arteries, as is shown in Figs. 94 and 95, from a case of aneurysm of the arch of the aorta.

The common causes of such local variations are abnormal



FIG. 95.—Tracing from left radial artery.

anatomical distribution, occlusion of vessels by embolism, thrombosis, sclerosis, or injury, and alteration by the pressure of tumours or aneurysms.

In certain conditions of the circulation recurrent pulsation is met with. When the radial artery is completely obliterated by the pressure of the finger placed nearest to the heart, there may yet be felt with the other fingers a feeble beat later in time than the pulse. The explanation of this phenomenon is that the wave of increased pressure has passed round from another artery through anastomosis; in the case of the radial artery it is from the ulnar by means of the palmar arch.

This symptom is not uncommon in febrile conditions, and more especially in pneumonia. In aortic regurgitation it is frequently found.

The characters of the pulsation to be met with over aneurysms of the aorta and great vessels will be discussed in a subsequent chapter.

*Blood Pressure in the Arteries.*—Attempts to estimate the blood pressure in man have been made by many observers. Marey, von Basch, Waldenburg, Hoorweg, Potain, Bloch, Mosso, Oliver, and Hill may be mentioned as having more particularly devoted their energies to the investigation of this point. The methods devised by von Basch, Oliver, and Hill are easy of application, and give results of real value. The plan adopted by Oliver in the construction of his pulse-gauge is to estimate the amount of arterial pressure by means of a spring connected with an index which records upon a dial pressures expressed in grammes. Oliver prefers this mode of stating pressure as simpler than the plan adopted by physiologists who gauge pressure in millimetre of mercury; but he has found that the two methods of estimating pressure very nearly coincide, the pressure of 105 grammes being equivalent to 100 millimetres of mercury.

The sphygmomanometer of von Basch consists of a pelotte which is applied to the artery whose pressure is to be investigated, and which is connected by a tube with a manometer. The manometer is constructed like the aneroid barometer, and consists of a box connected with the pelotte, the movements of which are communicated to an index travelling round a dial upon which the pressure is expressed in mm. Hg. Hill and Barnard have recently devised a very simple and wonderfully accurate form of sphygmometer which is specially adapted for clinical use. The pressure in this instrument is estimated by means of a metal tambour, the expansion of which is shown greatly magnified by means of an index travelling round a dial, graduated in millimetres of mercury. The tambour is connected by means of an indiarubber tube with a rubber bag attached to a flexible steel band, which is intended to be strapped round the arm of the adult or the thigh of the child. The connection between the pressure gauge and the

armlet is established by means of a Y-tube, to the third limb of which is attached a small compressing air pump fitted with a valve. The instrument is used by strapping the armlet round the arm, so that it fits closely to the skin, after which the pressure is raised within the rubber bag by means of the pump until the pulsation indicated by the index of the pressure gauge becomes of maximal excursion, at which point the pressure, as indicated by the gauge, is the mean arterial pressure. The reason why the point of maximal excursion is taken as that of the mean arterial pressure is that when the rubber bag is applied to the outside of the arterial wall with a pressure equal to that within the vessel, the walls of the artery are found to oscillate with the greatest freedom. Hill and Barnard have repeatedly proved the accuracy of this method by testing it with the neck of a dog while the femoral artery was connected with a mercurial manometer. They found that the normal arterial pressure in most healthy young men seems to lie between 110 and 130 mm. Hg. in the sitting posture. As the result of a considerable number of observations with this instrument, there is no doubt left in my own mind as to its simplicity and accuracy. In healthy young men the pressure has usually been from 105 to 135 mm. Hg. In cases of failing compensation in valvular disease it has varied between 60 and 70 mm. Hg. In cases of arterial sclerosis with renal cirrhosis it has been from 160 to 180 mm. Hg. My own observations do not yet entitle me to express very definite views in regard to the practical utility of the instrument, but there can be no doubt that it furnishes a most interesting addition to our methods of investigating the circulatory system.

*Auscultation of the Arteries.*—Auscultation of the larger arteries throughout the body reveals the existence of certain sounds. These sounds are subject to considerable variation.

It has been known since the observations of Weil that on auscultation of the large arteries situated near the heart, two distinct sounds can be heard in an overwhelming proportion of persons observed. The first of these sounds, which occurs almost simultaneously with the distension of the arteries, is weak in intensity and low in tone; the second, which ac-

companies the contraction of the blood vessel, is louder in sound and higher in pitch. The sounds are much the same, in their individual character and in their relations to each other, as those heard over the base of the heart, and they are simply the heart sounds propagated into the arteries. The fact might be regarded as sufficiently proved by the further observation that on listening over arteries far from the heart, as for instance the femoral, no sound is in most instances to be heard.

If the stethoscope is pressed more firmly upon the artery, the first of these sounds is accompanied by a murmur, and by pressing more firmly still the murmur may become so loud as almost to obliterate the sound. The second sound heard over the arteries is never in health accompanied by a murmur, no matter how much pressure be employed. The explanation of these appearances is quite clear. The pressure of the stethoscope on the artery produces a murmur at the point of constriction on account of the onward passage of the blood coincident with the first sound heard, but during the cardiac diastole or arterial systole there is not sufficient backward movement of the blood to give rise to any abnormal sound.

Under certain circumstances, as for instance in aortic regurgitation, the backward passage of the blood during the cardiac systole may be so great as to give rise to a murmur with the cardiac diastole as well as with the cardiac systole. This subject will be found more fully discussed in speaking of aortic incompetence.

THE CAPILLARIES.—Attention is elsewhere directed to the complexion as an indication of circulatory disturbance, and reference must be made in this place to the appearance commonly known as the capillary pulse. In many conditions of low arterial pressure an alternate flushing and pallor may be observed coincident with the rise and fall of the pressure wave. The symptom may be observed by examining the nails; more especially when a slight pressure is exerted upon the bulbous extremity of the finger, so as to produce a slight pallor towards the extremity of the nail; at the junction of the artificially produced pale portion and the normal pink tint of the nail elsewhere, the alternate waxing



and waning of the colour may be distinctly seen. The phenomenon may be observed even more distinctly by producing a flush over some part of the body normally pale, such as the forehead. On carefully examining such a patch the colour may be seen to increase and decrease synchronously with the movements of the heart and blood vessels.

The condition is seen very distinctly in aortic regurgitation. It is, however, by no means confined to this affection, as in all states of decreased blood pressure it may make its appearance. As will be seen in the section dealing with aortic and pulmonary lesions, its presence or absence is not without considerable diagnostic import.

The venous capillaries are in a condition of profound distension in cyanosis, as is elsewhere fully described.

THE VEINS.—On inspection, some of the peripheral veins



FIG. 96.—Tracing from vein on the back of the hand in a patient with acute pneumonia and aortic incompetence.

may be found to exhibit distinct movements, and such movements are most evident in thin persons, for all the superficial veins are less distinctly seen when the subcutaneous tissues contain abundance of fat.

In conditions of great relaxation of the arterioles, a direct venous pulse is sometimes to be observed in the veins of the extremities, particularly on the back of the hand and foot. Such a pulsation is obviously produced by the onward passage of the blood through the arterioles and capillaries into the venous radicles. The movement is sometimes sufficiently large to allow a tracing to be obtained, a good example of which is shown in Fig. 96. It was obtained from a patient suffering from acute pneumonia along with aortic incompetence, the full details of which will be found in the section devoted to the latter affection.

There are also appearances connected with the veins due to a retardation, instead of acceleration, of their movements. The movements of the veins of the neck under the influence of

respiration have long been known. Morgagni describes some experiments made by Valsalva which showed the swelling of the cervical veins during expiration and their subsidence during inspiration. Haller fully described this appearance, and Barry demonstrated the effects of respiration on the veins by tying the jugular vein, opening it on the central side of the ligature, and inserting the end of a curved glass tube, the other end of which dipped into a vessel of coloured fluid. He found that with each inspiration the fluid rose and on expiration it either remained stationary or sank a little. Poiseuille at a later period repeated these experiments, and found that in the jugular vein, with the valves intact, the only movement was towards the heart, but that when the tube was pushed past the mouth of the vessel into the innominate vein the retraction and repulsion of the fluid with inspiration and expiration was more nearly equal. In the physiological introduction the physical problems presented by the thorax have received full attention, and it is unnecessary to do more than recall one or two facts. There is less pressure on the blood contained in the vena cava and venæ anonymæ than on that of the veins of the neck; that is to say, the former are under less than atmospheric pressure, or, in other words, are under negative pressure. During the act of inspiration there is a constant lowering of pressure on all the contents of the thorax, and venous blood under atmospheric pressure flows in, as well as air, to equalise as far as possible the intra-thoracic and extra-thoracic pressures. Even during expiration the pressure is less upon the intra-thoracic veins than upon those of the neck, for the expiratory act caused partly by the mechanical arrangements of the thoracic walls, but more especially by the elastic retraction of the lungs, still leaves the lungs in a state of tension, as proved by their collapse on opening the thorax, as well as by the experiments of Donders. Although there is therefore at all times an inferior degree of pressure upon the vessels within the thorax, the difference is not so great during expiration as during inspiration. With forced expiration, however, the case is different. When the pressure upon the veins within the thorax rises to be equal to, or greater than, that on the vessels outside, or becomes positive, there is a tendency to backward flow. This

reflux, as pointed out by Hamernjk, closes the valves of the jugular veins, and, as long as the thoracic exceeds the atmospheric pressure, keeps them closed, so that the blood returning from the periphery accumulates above and causes a swelling of the vessels. Hamernjk maintained that in health and with ordinary respiration the valves at the lower end of the cervical veins were always shut on expiration, but this view is scarcely tenable. All experience is against it, and it has been ably combated by Immermann. Although absent in ordinary circumstances this closure of the venous valves, and consequently of the vessels, always occurs during forced expiration with dilated chambers and incompetence of the auriculo-ventricular valve on the right side of the heart, as well as in any affection lessening the elastic retraction of the lungs.

Venous pulsation in the neck was noticed and described by Galen, whose writings contained the only observations on this subject until the beginning of last century, when Homberg published some really interesting remarks upon it. Lancisi fully grasped the significance of venous pulsation, and Morgagni recognised that there were very often two distinct impulses in the venous pulsation, one produced by the contraction of the auricle and the other by that of the ventricle. From the date of Morgagni's observations almost every writer upon the circulation has dealt with the subject, and the most important contributions to it will be found summarised in a contribution made by myself to the subject some years ago. No real advance took place until the application of the graphic method to the movements of the veins in the neck by Bamberger, whose tracing showed a saddle-shaped curve. Bamberger was under the impression that the first shoulder was caused by the systole of the ventricle, while the second was due to the contraction of the papillary muscles. Geigel shortly afterwards described a presystolic followed by primary and secondary systolic elevations of the curve. The first named he considered as arising from a stowage wave, and the second and third respectively as of auricular and ventricular origin. In the same year Marey figured and described a venous tracing with two waves, auricular and ventricular. In a subsequent communication Geigel stated that his further observations lent no

support to his former conclusions, and that the first wave appeared to be caused by the contraction of the right auricle, while the second and third were ventricular. Friedreich published a long series of observations upon the venous pulse, and described its typical form as consisting of a primary wave produced by the systole of the right auricle, a large wave following it caused by the systole of the ventricle, and the third wave reflected from the interior of the heart in the same way as the diastolic wave in the radial tracings of aortic regurgitation, as is shown by Naumann.

Riegel distinguished between the normal or physiological venous pulse, diastolic and presystolic in its rhythm, and the pathological or presystolic and systolic pulsation. The former he regarded as being probably always present under ordinary circumstances, while the latter or positive systolic venous pulsation can only have its origin in incompetence of the tricuspid valve. Potain also expressed the view that pulsation was normally present in the jugular veins. Ringer and Sainsbury made some valuable observations on this subject. More recently Mackenzie has contributed some luminous observations upon this subject, and has with greater exactness than has heretofore been exhibited shown the relationship of the different events in the venous pulsation in the neck.

During its contraction the auricle not only drives blood forward into the ventricle, but when engorged it sends a stream also into the widened mouth of the vena cava, which produces the auricular wave. During its diastole the auricle receives a forward flow of blood from the veins, but as from distension of the right heart the tricuspid valve is incompetent, it also receives a stream of blood from the ventricle. After the auricular wave there is a fall in the venous pressure, producing in the veins a "negative wave." This "negative wave" is interrupted as soon as the auricle is filled, which naturally occurs earlier when there is much blood regurgitating through the tricuspid orifice. If this happens before the ventricular systole is completed the contracting ventricle forces the blood back through the auricle into the veins and constitutes another "positive wave" in the neck—the ventricular wave. At the time of the closure of the pulmonary valves the flow of blood



forward from the ventricle being stopped, but the passage back through the tricuspid orifice being still open, an acceleration of the blood through the tricuspid orifice takes place momentarily just before the ventricle passes into diastole, and this increase may sometimes be observable in tracings by a sudden elevation of the ventricular wave. When the ventricle passes into diastole the blood flows from the auricle and veins into the ventricle, thereby producing a second "negative wave."

This form of venous pulsation is by far the most frequent and may be present in all forms of heart engorgement, from such a slight degree as may appear compatible with almost perfect health to the most extreme form resulting from left-sided valvular disease. Usually, however, when there is extreme backward pressure from the left heart, the venous pulse becomes modified. Under such circumstances the relatively powerful right ventricle drives the blood backwards through the tricuspid orifice with so much force that the thin-walled auricle loses all power of impressing its independent action on the blood contained in its cavity. Hence in the veins there is but one large "positive wave" due to the ventricular systole, and one large "negative wave" due to the ventricular diastole, and thus is constituted the ventricular venous pulse. Such are the two forms of venous pulsation observed in the neck, the auricular often due to some transient dilatation and merely an exaggeration of a normal phenomenon, the ventricular always due to some organic disease.

The following tracings from the internal jugular vein of the right side in cases of well-marked tricuspid regurgitation are all of the auricular type. It will be seen that while they vary somewhat in form, they all present a number of features in common. The variations in form are partly due to the individual peculiarities of each case as regards the degree of regurgitation and the energy of the cardiac movements, but they are also produced in great part by extrinsic conditions resulting from the phases of respiration.

In Fig. 97 and Fig. 98 respiration was suspended while the tracings were obtained. In Fig. 99 the breathing was infrequent and shallow, while in the three remaining figures,

100, 101, and 102, the respiration was carried out in an ordinary way.

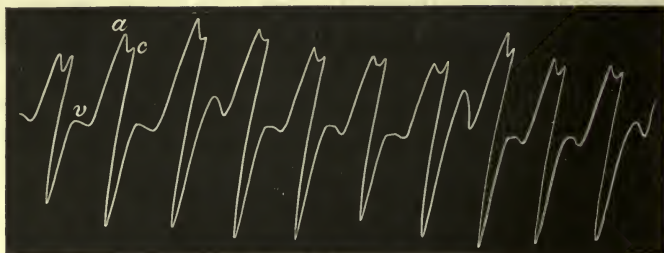


FIG. 97.—Tracing from internal jugular vein.

The respiration accounts for the larger curves in the trac-



FIG. 98.—Tracing from internal jugular vein.

ings, the line ascending during expiration owing to the filling



FIG. 99.—Tracing from internal jugular vein.

of the veins, and descending during inspiration owing to the emptying of them.

As regards the individual waves in each cycle of move-

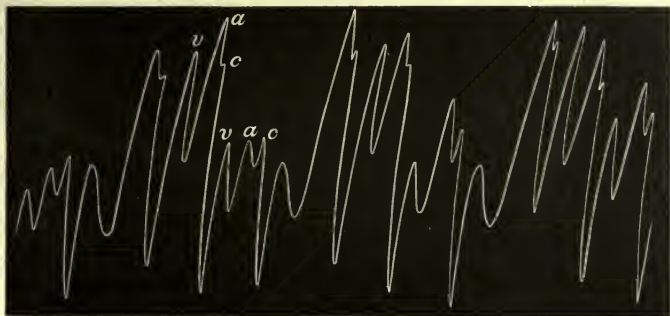


FIG. 100.—Tracing from internal jugular vein.

ment, these are marked in every case by letters denoting the causation of each wave.



FIG. 101.—Tracing from internal jugular vein.

The ventricular venous pulse when present in the neck is always found in the liver. It may, indeed, be absent from



FIG. 102.—Tracing from internal jugular vein.

the former while it is present in the latter. The auricular venous pulse when due to functional dilatation of the heart is

never present in the liver. When there is a venous pulse of the auricular type in the liver, there is organic valvular disease of the heart, and this is of necessity tricuspid stenosis. The reason for this is as follows. The ventricular venous pulse was assumed to arise in consequence of the distending and paralysing of the auricle by the forcible contraction of the ventricle. But if in place of a wide there is a narrow tricuspid orifice, the full force of the ventricle will be spent in forcing the blood through this narrowed orifice, and the tricuspid valves will stand between the ventricle and auricle, preventing the over-distension and paralysis of the latter, so that it continues to manifest its presence in the regurgitant stream of blood, and maintains in the veins the essential characters of the auricular venous pulse.

Auscultation of the veins of the neck furnishes several different kinds of sound, some of which may be regarded as physiological; others are mostly, if not entirely, met with in abnormal conditions.

The most important symptom furnished by auscultation of the veins is the continuous venous hum first observed by Laennec. He attributed its production to the arteries, although he mentions he had sometimes suspected that it was produced in the jugulars, and in this opinion he was followed by many other authorities who need not be mentioned in this connection. Ogier Ward was the first to suggest that the murmur was produced in the veins, and this view has gradually obtained universal assent. Aran demonstrated that the venous hum was more pronounced on the right than the left side of the neck, and that it could be accentuated by putting a strain upon the neck. He further showed that there might be a simple continuous murmur, or that it might be periodically reinforced by the action of the heart. Hamernjk, in his valuable contribution on vascular phenomena, elucidated the anatomical relations of the cervical veins with which the murmur is bound up. He showed that the internal jugular vein at the anterior root of the transverse process of the sixth cervical vertebra, or carotid tubercle, undergoes a considerable bend with its convexity directed forwards, that immediately below the carotid tubercle the vein is traversed by the omohyoid muscle, and that it could



be compressed and therefore narrowed by the action of the muscle. He further demonstrated that the vessel was wider on the right side in the triangular space, where it is simply surrounded by loose cellular and fatty tissue, but that on the left side it is more under the sterno-mastoid muscle so as to have firmer surroundings, while, at the same time, it can empty itself more easily into the innominate vein. This was a most valuable contribution to the elucidation of the subject. Hamernjk further suggested that the origin of the murmur was in vibrations of the venous walls, and that such a murmur could not be produced in any veins other than the jugulars.

Weil distinguished between continuous and intermittent venous hum, and subdivided the latter into four groups: (1) inspiratory; (2) diastolic; (3) inspiratory and diastolic; (4) continuous during inspiration and diastolic during expiration. Weil's most important contribution lies in the fact of his having pointed out that the venous hum is not significant of any particular condition of the circulation. His statistics agree with those of Wintrich in showing that the venous hum becomes progressively less common as age advances, and he found that there was little difference in the frequency of its occurrence when healthy persons were compared with those suffering from disease. With regard to the production of the hum, his views were clear that it took its origin in the passage of blood from a narrower portion of the vein into the relatively dilated jugular bulb.

The different views in explanation of the venous hum have been dismissed with the exception of that which regards the murmur as due to the production of audible vibrations by the blood in its passage from a narrower into a wider portion of the venous channel. As the veins accommodate themselves very closely to the amount of blood which they contain, it follows that if there be any reduction in the amount of blood the vein will contract in accordance with the quantity present, but as the vein when passing through the cervical fascia is kept wide by its attachments, it follows that although it may be narrower above it will remain widely open as it passes through the fascia. Weber and Čejka found that a venous hum could be detected in the femoral vein, either by

elevating the foot or by gently compressing the vessels in the upper portion of the thigh.

The venous murmur now under discussion is usually continuous, that is to say, it is present during every phase of the cardiac cycle. Although continuous it is by no means uniform in its intensity, since it waxes and wanes, not only with the systole and diastole of the heart, but also with the movements of respiration. It is found to be louder during or immediately after the diastole of the auricle, and it is also louder during inspiration. In its character it presents very considerable differences, being in some instances soft and sighing, while in other cases it is harsh and roaring. This cervical murmur is best heard between the two attachments of the sterno-mastoid muscle. It is usually heard without any excess of pressure on the stethoscope, and in every position of the patient's neck. As a rule it is much louder when the patient sits up than in the recumbent posture, and in truth it very often cannot be found in the latter position. It is sometimes better heard when the patient turns the head to the opposite side from that which is being examined. As a rule it is louder upon the right side, but this is not universal. It is not uncommon to find that this continuous murmur is heard over the manubrium sterni; it may, indeed, be traced down in suitable instances as far as the aortic area.

A valvular sound was discovered by von Bamberger in the jugular veins. He found that a distinct but dull sound occurred with the cardiac systole in cases of tricuspid regurgitation, and regarded it as produced by tension of the venous valves in consequence of the reflux. This sound can occasionally be made out under such conditions, but it is not to be regarded as at all common in its occurrence. Such sounds may also be heard in the femoral veins, and, as was pointed out by Friedreich, they may be double, which he attributed to reflux waves from the auricle as well as from the ventricle. These venous sounds occasionally pass into murmurs both in the jugular and femoral region, and when such is the case it probably speaks for incompetence of the venous valves. Since the normal condition of these valves, however, is a matter that cannot be regarded as definitely settled, it would

certainly be unwise to claim any diagnostic significance for this interesting clinical fact.

This concludes the consideration of the different effects produced upon the peripheral circulation by cardiac and vascular disease, but certain of the appearances already reviewed cannot fail to present themselves for further study in the next section.

#### SYMPTOMS CONNECTED WITH OTHER SYSTEMS.

The extensive and intimate relations of the circulation with every function of the body render it inevitable that any derangement of the processes which it maintains must be followed by consequences of the widest range. What the general pathological effects are has already been shown; there still remain for consideration and explanation, however, the various clinical appearances to which they give rise.

In analysing the symptoms of the diseases of the circulation, the most satisfactory method is to group them according to the physiological systems which are affected. The various clinical features, therefore, will be considered according as they are digestive, hæmopoietic, respiratory, cutaneous, urinary, reproductive, or nervous.

SYMPTOMS CONNECTED WITH THE DIGESTIVE SYSTEM.—The functions of digestion are frequently disordered in circulatory affections. In venous stasis there are almost always chronic catarrh and passive congestion of the mucous membrane. These are revealed by nausea, vomiting, and diarrhœa, as well as by hæmatemesis and melæna. The tongue is commonly found in such cases to be flabby and coated, while the hæmorrhoidal veins may be dilated. In such conditions the stomach and intestines are distended on account of loss of tone, and lead to a degree of meteorism which is not only troublesome but often in many instances dangerous.

Jaundice is a common symptom of cardiac disease, and may even attain considerable intensity. It is obviously produced by the interference through venous stasis with the return of blood by the hepatic vein. In the presence of backward pressure the liver may present different symptoms. It may be simply enlarged on account of the venous stasis to which it is subjected, or it may manifest pulsation. The

pulsation is usually found to consist in a simple impulse communicated by the movements of the heart. It has commonly been taught that the impulse follows the systole of the ventricles. This, however, is not the case, and my own observations entirely agree with the views of Mackenzie that the impulse follows, and is produced by the diastole. There is also, however, another form of liver pulsation which really follows the systole of the ventricles, and is produced by a reflux of blood into the inferior vena cava. The liver, moreover, shows a pulsation preceding that of the ventricle, giving, therefore, the features of the auricular pulsation of Mackenzie, who has called attention to this particular type of pulsation as a probable indication of tricuspid obstruction. This will be more fully dealt with in connection with that affection.

In consequence of the venous stasis, transudation from the peritoneum frequently takes place, and ascites is, therefore, a common symptom in cardiac disease. This subject, however, will be discussed in connection with the serous membranes.

**SYMPTOMS CONNECTED WITH THE HÆMOPOIETIC SYSTEM.**—Several important symptoms are elicited on investigating the condition of the blood and the glands. Some of these are apparently linked with circulatory disorders by a direct causal nexus, that is to say, the factors present are operative as regards the production both of a change in the blood and at the same time an alteration in the circulatory mechanism. Others, however, appear to take their origin directly in the circulatory disturbance, of which they, therefore, are regarded as symptomatic.

*The Blood.*—When *anæmia* is present along with cardiac disease, it may simply present its usual features as regards the reduction of the quantity of hæmoglobin and of the number of the red blood corpuscles, along with, in some cases an increase, in others a decrease, in the number of the white blood corpuscles.

In *cyanosis*, noteworthy changes are found in the blood. It is not only dark coloured to the naked eye, but on investigation it shows some striking alterations. The specific gravity is raised. The amount of hæmoglobin undergoes a remarkable increase, and the number both of the coloured and colourless corpuscles is greatly augmented, as seems to have been first noticed by Toeniessen, whose observations have been frequently



repeated subsequently—for instance, by Penzoldt, Banholzer, Krehl, Vaquez, and Carmichael. The specific gravity has been found as high as from 1070 to 1080. The quantity of hæmoglobin in one of Banholzer's cases was 160 per cent. The number of red blood corpuscles has frequently been found to exceed 9,000,000 per cm., and the number of white blood corpuscles has reached 16,000 per cm.

Starting with the conception that cyanosis is produced by obstruction to the circulation and consequent venous stasis, the question now arises why this condition should be associated with an increase in the number of the blood corpuscles. It is not only in the cyanosis of congenital lesions that the increase is found, but in all cases where cyanosis is present on account of failure of the circulation, whether primary or secondary. To this point Toeniessen and Schneider have particularly called attention, and of the accuracy of the observation any one may be convinced by investigation of the blood. The work of Malassez seems to show that the blood in the superficial parts of the body contains a larger number of red corpuscles than that from the deeper layers, and Penzoldt and Toeniessen believe that this increase is caused by the loss of fluid from the surface, while the blood of the interior is constantly receiving fluid from the alimentary canal. Even if this be correct, it cannot be accepted as an explanation of the great increase in the number of the corpuscles found in cyanosis; it would involve the postulate that in some cases, where the number of corpuscles is doubled, only half of the usual quantity of the fluids of the blood must be retained within the blood vessels. Cohnheim's celebrated experiment of tying the crural vein of the frog, which is followed by a considerable increase of the corpuscles in the vessels, with the transudation of serum into the surrounding tissues, may be regarded as an explanation of the moderate increase in the cases accompanied by anasarca, but it has no special bearing upon cases of congenital cyanosis in which there is no drain of fluid into the tissues. It may possibly be held that in such cases the lymph vessels are unusually active, and that the fluid constituents of the blood are as rapidly absorbed as they transude. Such an opinion can scarcely be seriously entertained. The backward pressure

on the venous system which causes the transudation must tell on both terminations of the absorbents. It may be admitted freely that the increased pressure on the peripheral veins may tend to raise that impelling the fluids into the commencement of the lymphatics, but it must not be forgotten that an elevation of pressure in the great veins will hinder the return of the lymph by pressing upon the openings of the lymph vessels into the veins. It is probable that the increase in the red corpuscles may be to some extent compensatory in cases of cyanosis. To say this, however, is not enough; it affords no rational explanation of the process by which the increase is brought about. Nature does not work by such direct methods as would require to be invoked if the increase of the corpuscles were regarded as a simple compensatory change, balancing the diminished power of oxygenation. Compensation in valvular lesions, for example, is produced by the definite structural changes constituting hypertrophy, caused by increase of work, and compensation in cyanosis must have some reasonable explanation also. An attempt at such an explanation was recently made by me from a consideration of the functions of the red corpuscles. In venous stasis the corpuscles are insufficiently oxygenated, they cannot thoroughly perform their duties as oxygen carriers, and they cannot yield so much oxygen to the tissues. It must further be remembered that in cyanosis there is less metabolism in the tissues, and therefore less waste produced. In a word, the functions of the corpuscles being lessened, the tear and wear which they undergo is reduced, and the duration of their individual existence increased. The number of the corpuscles must in this way be proportionately augmented, and this must lead to the numerical increase, as well as to the high percentage of hæmoglobin, until a balance is struck between the production and the destruction of the blood corpuscles.

Marie has recently attempted to draw a parallel between the well-known observations of Bard and Curtillet, Viault, Grawitz, Zuntz, Vaquez, Mercier, and others, on the state of the blood at high altitudes, and its condition in cyanosis. The number of red blood corpuscles is subject to considerable augmentation in higher levels, and the increase appears to

be proportional to the altitude. Marie gives, for example, the following table illustrative of these facts:—

Christiania (sea-level)	.	Number of red blood corpuscles,	4,970,000
Göttingen (148 metres)	.	" "	5,225,000
Tübingen (314 metres)	.	" "	5,322,000
Zürich (412 metres)	.	" "	5,752,000
Görbersdorf (461 metres).		" "	5,800,000
Reiboldsgrün (700 metres)		" "	5,970,000
Arosa (1800 metres)	.	" "	7,000,000
Cordilleras (4382 metres)		" "	8,000,000

He is of opinion that on account of the rarefaction of the air and of the diminution of atmospheric pressure, there is some difficulty in hæmotosis, and that in order to overcome the difficulty, the means employed is an increase in the number of the red corpuscles and in the amount of hæmoglobin. Marie makes no attempt to explain how such a compensatory change is effected, but it seems to me that my explanation of the increased number of red corpuscles and the augmented quantity of hæmoglobin is also valid for the polycythæmia of high altitudes. One word of caution may be expressed: we are not as yet in a position to state whether the increase in the number of red blood corpuscles and in the quantity of hæmoglobin is general or is purely confined to the superficial parts of the body, and until the facts have been ascertained it is idle to speculate further on the subject.

*The Glands.*—The *spleen* presents changes in size which are mostly dependent upon the state of the portal circulation. When there is much venous stasis with enlargement of the liver, the spleen almost invariably undergoes enlargement; but never, as a rule, to any great extent.

As the concomitant of other conditions accidentally associated with circulatory disorders, the *thyroid gland* may be enlarged or diminished. Enlargement of the thyroid gland as part of the complex of symptoms known as Graves' disease is sometimes found in association with organic disease of the heart. Regarding this affection as one in which the circulatory disturbances are secondary, it is not in my opinion expedient to discuss it in this work, but frequent reference must be made to it.

## SYMPTOMS CONNECTED WITH THE RESPIRATORY SYSTEM.—

There are some symptoms connected with respiration of much importance in estimating the gravity of circulatory disturbances. These symptoms may have their origin in conditions other than circulatory disturbances, and there is, therefore, nothing pathognomonic about them. They require the most rigid scrutiny, in order to ascertain their origin; only when this has been done can they be regarded as giving useful evidence with regard to the affections of the circulation. The breathing may undergo several distinct changes, all of which are interesting in themselves, but only some of these have relation to the state of the circulation. It is, therefore, necessary in the following pages to restrict the subject to those aspects of respiratory alteration which arise from changes in the circulation.

*Dyspnœa*.—Breathlessness presents several different clinical types, and has many different causal factors. It may consist merely in breath hurry, or polypnœa; it may become laboured and distressful, when it is strictly speaking known as dyspnœa; and when it is only possible for the patient to breathe in an upright position it is commonly known as orthopnœa.

Breathlessness occurs sometimes only on exertion; it sometimes presents the features of recurring paroxysms; it is sometimes present continuously without intermission.

The essential cause of breathlessness is deficiency of oxygen in the blood supplying the respiratory centres. It is universally recognised that there are differences in the kind of breathlessness produced by mere deficiency of oxygen, or excess of carbonic acid. In the case of a simple deficiency of oxygen, the dyspnœa is much more intense and enduring than when there is excess of carbonic acid; in this latter case the poisonous influence of the carbonic acid soon brings the respiratory movements to an end. It is well known that disease in, or pressure upon, the nerve centres connected with the respiratory processes may produce dyspnœa. With such facts we have, however, no immediate concern in this connection, and attention must be solely concentrated upon the dyspnœa which results from interference with the blood supply to the centres. The blood supply to the centres may fail in



respect of alterations in the driving power of the heart, of the conduction possibilities of the blood vessels, or the nutritive capabilities of the blood itself.

The heart may simply be inadequate to the duties required of it, so that the least relative over-exertion by muscular effort or mental emotion will give rise to breathlessness. This is particularly the case in those who, on account of obesity, have already some mechanical hindrances to respiratory processes. This is still more the case when there is some transient cause, such as the cardiac debility or myocarditis of pyrexia, or such a permanent factor as any degenerative process connected with the heart muscle. In certain instances of disease, such as anæmia, there is debility of the heart wall, as well as impaired oxygenating possibility on the part of the blood. In arterio-sclerosis the interference with the vascular supply is usually linked with chronic myocarditis.

Certain of the valvular lesions are particularly prone to cause breathlessness, more especially incompetence of the mitral valve. Such lesions, by diminishing either the suction or the force-pump action of the heart, or both of these, may give rise to passive congestion and œdema of the lungs, and to hydrothorax. When the venous stasis involves the right side of the heart and produces its effects upon the liver, a new train of symptoms is developed, the result of which is to deepen the respiratory distress, since passive congestion of the stomach and intestines interferes with the free movements of the diaphragm, and if ascites be superadded the disturbance is proportionately increased.

It is quite possible that, when there is a cyanotic condition, or, even short of this, when there is simply a feeble circulation, there may be an excitement of such cortical centres as have been suggested by Christiani; and that these, by sending impulses downwards to the mechanical centres for respiration in the medulla and spinal cord, produce dyspnoea.

It is well known that in almost all feverish conditions there is acceleration of the respiration. There are probably two distinct causes at work in pyrexia. On the one hand, the increased metabolic functions give rise to the presence of an excess of waste products in the blood; on the other hand,

the higher temperature of the blood must have some effect, since, as is well known, when the blood of the carotid arteries is heated in any animal dyspnœa is produced.

There is always dyspnœa in anæmia, the reason for which is obvious. The reduction in the amount of hæmoglobin lessens the possibilities of oxygen supply to the centres, while at the same time they are probably in a condition of irritable weakness from malnutrition. There must ever be borne in mind, however, the consideration that, in addition to such factors, the feeble condition of the heart muscle plays an important part in the production of dyspnœa, more particularly in the dyspnœa of exertion.

The paroxysmal dyspnœa of circulatory affections, often termed cardiac asthma, is undoubtedly due to failure of the blood supply to the nerve centres. This much is clear, but we have no accepted theory explaining it. It seems to me, nevertheless, that its explanation cannot be a matter of great difficulty, as will be more fully mentioned in the succeeding section on Cheyne-Stokes' breathing. When the nerve centres are in a state of impaired nutrition they have a tendency to undergo modifications in the rhythm of their functions, and to my mind cardiac asthma is, in its recurrent, often periodic, phases, strictly analogous to the appearances in Cheyne-Stokes' respiration. The only explanation for it lies in the undeniable tendency of structures manifesting rhythmic activity to have the periodic manifestations of this energy interfered with by the imposition of a larger secondary rhythm upon that which is primary and inherent.

Cardiac asthma is marked by a total absence of expiratory dyspnœa. It is believed by Sée to be caused by a voluminous condition of the lungs from stasis. Whether this be invariably the case or not, there can be no doubt as to its frequent association with arterial sclerosis, high blood pressure, and slowing of the blood current.

It is not at all uncommon to have certain blood changes associated with circulatory disturbances. The uræmia of renal disease, and the glycæmia of diabetes mellitus present the best examples of such blood impurities, and it is well known that these give well-marked examples of dyspnœa.

Dyspnœa may therefore be considered as having its origin in some instances from simple failure of blood pressure rendering it impossible for the blood to be sufficiently oxygenated in its passage through the lungs.

Such an interference with the respiratory functions must of necessity be increased if there be any catarrhal condition of the mucous membrane in the smaller bronchial tubes, and as retrograde effects upon the bronchial veins result from increased pressure in the right chambers of the heart, any alteration on that side is apt to evoke stasis and catarrh. Bronchial catarrh makes its presence known by more or less characteristic physical signs. There may sometimes be nothing more than prolongation of expiration with a harsh character of the breath sound, or there may on the other hand be not only sonorous or sibilant rhonchi from the catarrhal change in the tubes, but there may also be some moist sounds in consequence of some serous transudation into them.

Passive hyperæmia leading to œdema of the lungs takes its origin in any increased pressure in the left auricle, and these conditions are a fertile source of dyspnœa. Congestion and œdema are not revealed by inspection, but on palpation there may be a slight increase in the vocal fremitus, and on percussion a slight degree of dulness may be elicited. On auscultation there may be nothing more than some roughening of the respiratory murmur and the presence of moist sounds, usually in the form of fine crepitations, along with a slight increase in the intensity of the vocal resonance.

Dyspnœa takes its origin also in hydrothorax. When much fluid is present in the pleural sacs there is almost invariably orthopnœa. The physical signs yielded by hydrothorax require in most instances to be sought for, and as it is one of the most common results of left-sided heart disease, the examination of the patient with a view to its detection is a duty which cannot be too strongly insisted upon. Sometimes on inspection there is an obvious enlargement of the lower part of the chest posteriorly, and a bulging of the intercostal spaces is even sometimes described. It must, however, be somewhat uncommon. On palpation the vocal fremitus is greatly diminished or entirely absent over the affected region.

On percussion there is a great degree of dulness, and on auscultation the respiratory murmur and the voice sounds are almost or quite suppressed. On withdrawing some of the fluid by means of the aspirator it is usually found to be semi-transparent and of a pale yellowish green colour. Its physical and chemical characters, as well as the differences between a transudation and exudation, have been already referred to.

*Periodic Respiration.*—Cheyne-Stokes' respiration is the term employed to denote a periodic form of respiratory rhythm, possibly noted by Hippocrates, and certainly observed by Hunter, as well as by Nicolas, who is mentioned by Gallais, but first brought into prominence by the two physicians with whose names it is linked. It does not necessarily include any irregular arrests of breathing, such as are frequently observed in diseases

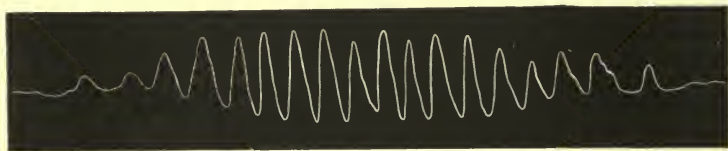


FIG. 103.—Tracing of Cheyne-Stokes' breathing.

of the brain, and which are generally classed together under the term cerebral breathing, but there is no hard and fast line between the regular periodicity of events in Cheyne-Stokes' breathing and the irregular interruptions of breathing in cerebral respiration. So many intermediate links are to be found between the two extremes that the existence of an uninterrupted series of symptoms may safely be assumed.

In Cheyne-Stokes' breathing the normal rhythm is interrupted by distinct arrests of respiration; there is an alternation of periods of activity and periods of repose. The arrests of respiration occur periodically, and it may be said that there is a secondary, as it were, grafted upon the primary, rhythm of breathing. This, however, is not all, for an essential feature is that the period of activity consists of two distinct phases in which the extent of the movements waxes and wanes. During the former of these there is a gradual increase not only in the amplitude of the respiratory movements, but also in their rate, while during the latter phase there is a gradual decrease, both



in the extent and rate of these movements. A tracing of the thoracic movements is annexed (Fig. 103).

Cheyne-Stokes' respiration is frequently associated with other symptoms. There are, first, alterations in the state of the circulation, originally observed by Reid. These are at once inconstant and variable, contrasting markedly with the regularity of the respiratory phenomena. Sometimes the rate of the pulse is diminished during the pause in respiration, but on the other hand it has been observed to be greater during the period of repose than during the period of breathing. Variations also in the volume and pressure of the pulse have been met with, and it has been described as of larger volume and lower pressure during the arrest of breathing than during the period of activity. In many cases no periodic change in the state of the circulation can be observed on the closest investigation.

There are also certain appearances connected with the eyes which sometimes occur in association with Cheyne-Stokes' breathing. In many cases the periodic rhythm of the respiration only takes place during sleep, when, as may readily be understood, no changes are observed in the eye, but in a large number of instances the symptom is present during the waking as well as the sleeping hours. In some of these latter cases, as was noticed by Leube, the eyes opened during the period of breathing and closed during the period of respiration. During the breathing, further, the pupil is dilated, and during the pause it is contracted. As Leube pointed out, the dilatation begins along with the early superficial respirations, or may even precede the active phase of the breathing cycle. Appearances exactly similar in character were observed by Leyden as a result of experiments on the medulla. It has also been noticed by Finlayson that the pupil dilates a little with each inspiration, and contracts slightly with each expiration, until the height of the respiratory phase is attained, when the pupil is widely dilated, after which the contraction with each expiration somewhat exceeds the dilatation accompanying each inspiration, until at the end of the active phase the pupil becomes fixed in the contracted condition. As Merkel observed, the pupil reflex is absolutely abolished during

the cessation of respiration, and no reaction to light can be elicited. No changes in the vessels of the retina have been seen during the varying phases of respiration, as has been clearly determined by Schepelern and Ewald.

The condition of the mental powers undergoes fluctuations in Cheyne-Stokes' respiration. The most usual state of matters is, as Leube showed, that the patient is conscious during the period of breathing, while during the interval of repose he sinks into unconsciousness.

A large number of explanations have been advanced to account for the altered rhythm of Cheyne-Stokes' breathing, most of which have been discussed by me in a previous work. It is clearly impossible in this place to do more than indicate the most important views possessing any degree of scientific probability, leaving without comment those which cannot be looked upon as in any respect worthy of attention.

The theories fall naturally into two groups. In the first place there have been attempts to explain the regular periodicity of the breathing by varying conditions of the stimuli acting upon the nervous mechanism of respiration.

The theory of Traube rests upon the conception of a lessened supply of arterial blood to the medulla, in consequence of which the respiratory centre is supplied with less oxygen. Through this lessened amount of oxygen the irritability of the nerve cells becomes lowered and a larger quantity of carbonic acid is required to cause an inspiration; the time, therefore, within which the acid will accumulate in sufficient quantity is lengthened. The lessened irritability of the centre requires, however, a larger amount of carbonic acid as a stimulant, and thus there is a long pause. When this gas has accumulated in sufficient quantity it first stimulates the pulmonary terminations of the vagi, but the strongest stimuli applied to these nerves never cause dyspnœa, and this only produces the shallow breathing first appearing after the pause. The amount of carbonic acid further increases sufficiently to cause stimulation of the nerves coming from the skin and other parts of the body, and hence the forcible breathing sets in. The quantity of the gas is in turn greatly diminished by the dyspnœa, and the excitement of the other

nerves ceases, so with the action of the vagi alone shallow breathing again occurs until there is not enough carbonic acid gas to excite the pulmonary endings of the vagi, and the pause sets in anew.

A simple and constant reduction of the functional activity of the respiratory centre could not by any possibility induce a change from regular rhythm to periodic rhythm of the respiratory movements; under trenchant criticism, indeed, Traube found himself obliged to shift his ground, and in restating his theory he fell back upon a tendency to rhythmic periodicity in the respiratory centre.

The explanation of Filehne admits that there must be a lowering of the irritability of the respiratory centre, but asserts that the irritability of this centre must be reduced in a greater degree than that of the vasomotor centre. He asserts that in health venous blood excites in regular order (1st) the respiratory, (2nd) the vasomotor, and (3rd) the convulsive centres. When the Cheyne-Stokes' phenomenon is present, the blood during the pause gradually becomes more venous and develops the stimulus for the centres, but, from the lessened irritability of the respiratory centre, no respiration is caused, and the pause therefore continues until the point is reached when the vasomotor centre is brought into action. This produces a diminution of the blood supply, which causes the respiratory centre to act and originate the superficial breathing which is first observed. Some time, however, elapses before the blood arterialised by these respirations can reach the vasomotor centre, and this is delayed by the contraction of the arterioles caused by its activity; it also takes time before the vasomotor apparatus can induce contraction of the arterioles, and time also before the contraction can pass away; there is therefore a lengthening of the pause and deepening of the dyspnœa.

The views of Filehne have been adopted by Bramwell with certain modifications, more particularly in regard to the possibility of a different state of excitability in the discharging and restraining portions of the centre, and with regard to a state of irritable weakness rendering the centre more easily fatigued and yet also producing more powerful effects when sufficiently stimulated.

Such complicated hypotheses rest on assumptions as yet unproved, and on appearances at least inconstant. The statements in regard to the excitability of the centres in the medulla rest upon no basis of fact, and, with reference to an arterial spasm, upon a misconception. Filehne's observations on the alterations of pulse rate of pressure have been found by numerous writers to be altogether incorrect, inasmuch as the changes when present at all have been seen to be the opposite of what he described, and to occur at a period entirely different to that stated by him. An arterial spasm, moreover, in a condition of lessened cerebral activity, would probably lead to consequences quite the opposite of those seen at the beginning of the phase of breathing. His observations finally on the effect of amyl nitrite in Cheyne-Stokes' breathing have been refuted by myself.

In the second place are theories which seek to explain the regular periodicity of the respiration by variations in the intrinsic condition of the respiratory centre.

Observing that in certain circumstances the contractions of the heart of the frog fell into groups or became periodic, Luciani was led by the resemblance of this phenomenon to the character of Cheyne-Stokes' breathing, to seek for some condition common to both as a cause of the two phenomena. He does not think that Cheyne-Stokes' breathing can be explained by means of the principle, that the capacity and activity of a nervous organ depend on extrinsic stimulant and nutritive conditions. No doubt the vitality of any organ is intimately connected with surrounding conditions and influences, but it does not follow that the organ in every case transforms only as much as it receives in the same measure, as well as in the same rhythm, with which it receives it. Drawing a clear line of distinction between reflex and automatic movements, Luciani points out that the determining cause of the former is extrinsic, while in the case of the latter it is intrinsic, and consists in oscillations of the internal nutritive movements, to which correspond as many oscillations of the excitability of the organ itself. He was led to this conception of automatism by the discovery of the periodic grouping of the movements of the frog's heart, for it could not be doubted that when



extrinsic conditions remained unchanged, the cause of the alternate groups of pulsations and pauses was intrinsic. Luciani therefore regards the diverse forms of respiratory rhythm as extrinsic expressions of the nutritive changes in the structure of the respiratory centre. If it be granted that the respiratory centre is automatic, it follows that the different forms of rhythm which constitute Cheyne-Stokes' phenomenon may be regarded as effects of diverse kinds of automatic oscillations in the excitability of the centre itself.

This theory presents us with views entirely different from any which preceded it. It is no doubt true that Traube in his corrected hypothesis makes mention of a tendency to rhythmic periodicity in the respiratory centre, but clearly indicates that this tendency is conditioned by extrinsic agencies. Luciani therefore makes quite a new departure in advancing the view that the change from regular to periodic rhythm is due to intrinsic conditions. The only difficulty as regards the theory of Luciani concerns the question whether the respiratory centre can be regarded as really automatic. As this is now universally granted, it must of necessity follow, that, although it is perhaps more influenced by external agencies than any similar organ, the nerve centre mainly concerned in the maintenance of respiration can modify its functions independently of such extrinsic conditions. There can therefore be no difficulty in accepting the view of Luciani.

Rosenbach finds an explanation of Cheyne-Stokes' breathing, with its attendant symptoms, in the natural alternation of activity and repose. In the respiration we see inspiration, expiration, and pause; in the circulation, systole, diastole, and pause; in the nervous system, waking and sleep; while in curarised animals there are periodic changes in the rate and pressure of the circulation which are quite independent of the respiration. The origin of activity is in the cell, not in the blood, and it is illogical to seek a cause of respiratory and other phenomena in the latter. Periodic activity of all nervous apparatus, therefore, depends on immanent peculiarities of elementary structures; the blood is not the direct stimulus for the cells, but exerts its power by giving the cells the possibility of regulating tissue change. When the blood is

altered there is necessarily a modification in the absorption of oxygen and the removal of the products of tissue change, and the mechanism will therefore be indirectly affected; the blood is thus only one link in the chain of apparatus needful for life. The regular alternation of activity and repose characteristic of life is seen in the complex of pathological phenomena, of which periodic breathing is only one symptom; Cheyne-Stokes' respiration is therefore the result of a condition in which the exhaustibility of the central apparatus, normally following its activity, is greatly increased. The respiratory centre has its irritability lowered, as the breathing is at first shallow, but the irritability progressively increases, for in spite of better aeration, dyspnoea gradually develops. The irritability then diminishes, and the descending phase begins.

The phenomenon, therefore, according to Rosenbach, occurs in conditions of disturbed nutrition, but these are independent of any periodicity in the blood supply to the brain, and are co-ordinated by, and joint effects of, one and the same cause occurring periodically in the central organs, this cause being exhaustion of the centres. The whole brain may be affected, when the entire complex of symptoms termed Cheyne-Stokes' phenomenon is produced, or limited tracts only may be implicated, giving Cheyne-Stokes' breathing. He points out that just as the respiratory centre alone may be deranged, so the vasomotor or vagus centre may be disturbed, as in tubercular meningitis, and cause changes in the tension or rate of the pulse. Rosenbach compares the periodic exhaustion with the normal pauses for rest shown by all rhythmically acting systems. The different phases resemble natural phenomena, but with longer intervals; the period of breathing, for example, is to be compared with a respiration, and the period of repose with the short pause following expiration. The vagus and vasomotor centres show similar variations. The exhaustion of the brain induces sleep, during which the pupils behave as in ordinary slumber. The centres are not only more easily exhausted, requiring longer rest, but their irritability is reduced, and forced breathing comes on in spite of better arterialisation of the blood (which involves reduction of stimulus). The meaning of this is, that the centre is becoming more irritable,

although the stimulus is lessening. After a time the normal irritability is regained, which is accompanied by gentler breathing until the pause resulting from exhaustion occurs. This theory runs on the lines laid down by Luciani, and assumes variations in the intrinsic condition of the centres as the cause of the periodicity of the phenomena.

The complex movements of respiration afford one of the most beautiful examples of co-ordinate muscular processes. The precision with which the different movements of the face, throat, larynx, and thorax are brought into harmony points to highly specialised nervous mechanism. The movements are not merely complementary to each other under ordinary conditions, but they may, in altered circumstances, become also compensatory. If the phrenic nerves, for example, are in any way inadequate, so that the diaphragm is incompetent, the intercostal muscles act more powerfully by way of compensation. Such facts prove that the respiratory centres have the power of increasing the activity of certain muscles in order to supply what is lacking on account of defective activity elsewhere. It is clear from the results of experiment that at least in young animals there are spinal as well as bulbar centres engaged in respiration, and this is analogous to the thoroughly established fact that there are vasomotor centres in the spinal cord as well as in the bowel. In both cases there are probably lower spinal, under the control of higher bulbar, centres.

The nervous mechanism of respiration is subject to the influence of external agencies, and the movements of respiration may be altered by processes taking place in the higher, as well as in the lower nervous tracts. The functions of the respiratory centre are also modified by changes in the blood circulating through the nervous textures. Venous blood augments the respiratory movements. Even after section of the vagi and of the spinal cord below the medulla, a venous state of the blood increases the facial respiratory movements. A diminished supply of blood produces increased respiratory efforts, and if the blood flowing upwards to the brain be heated artificially a similar result is obtained. Apnoea may be produced by forced respiration, but this cannot be caused so easily after section of the vagi.

Many phenomena produced by external agencies are purely reflex, but, after admitting this, there are other facts which point to something more than simple reflex action in the respiratory centre. In section of the vagi, for example, the respiration is maintained although considerably modified by the lack of the ascending regulative influence of these nerves, which proves that the movements of respiration are independent of, though modified by, impulses from the lungs. If the spinal cord be severed from the medulla, and all the accessible sensory cranial nerves be divided, so that the respiratory centre is freed from almost all sensory impulses, the respiratory movements of the face and larynx continue, although the thoracic movements necessarily cease. This observation proves that the respiratory movements are independent of all sensory stimuli, and that the respiratory centre is in its nature truly automatic. It must therefore be concluded that the centre is endowed with independent automatism, although subject to modifications of its activity by impulses from without.

Mosso has shown that in man and lower animals, perfectly healthy, but fatigued by exertion, breathing becomes periodic during sleep, an observation which has been verified by subsequent investigations, and which may be confirmed by any one who has doubts on the subject. Periodic breathing may therefore be regarded as in many instances a perfectly physiological appearance. In hibernating animals, as Mosso pointed out, the breathing is very similar to Cheyne-Stokes' respiration.

In the diverse forms of disease presenting Cheyne-Stokes' breathing one condition must be held to be constant—a reduction of the activity of the higher nervous structures. Whether the cause be in these centres primarily, or in other structures, such as the circulatory, there is the one constant condition of lowered nerve activity such as normally occurs in deep sleep, or after a full narcotic has been administered. The explanation of this phenomenon is supplied by the investigations of Marckwald, from which he has been led to conclude that periodic breathing can only occur when some of the higher brain tracts have ceased to exert their influence



upon the respiratory centre. During sleep the action of these higher tracts is in abeyance to a greater or lesser extent. In certain individuals a greater degree may habitually be present, in others it only takes place after great fatigue. The effects of many drugs which produce periodic breathing amply confirm this view, as all of them which have been found to produce such effects have the power of lessening cerebral activity. It is, however, extremely probable that in addition to the removal of the higher influences the activity of the respiratory centre itself must be lessened.

The periodic movements of respiration in hibernation were seen by Mosso to become regularly rhythmic on elevation of the external temperature, and periodicity of respiratory rhythm was caused by Fano in the alligator, by subjecting it to the influence of cold. It is a reasonable inference that in these instances the depressing influence of the low temperature lowers the vitality of the animal, and diminishes the control of the higher nervous centres, while it no doubt lessens the activity of the respiratory centres.

Such periodic phenomena are not confined to the respiration. Luciani has observed analogous phenomena in the amphibian heart when removed from its normal nervous control. Waller and Reid have seen similar appearances in the excised mammalian heart. Fano and Langendorff and Siebert have described corresponding changes in the heart removed from the embryo, and Solokow and Luchsinger in the lymph heart of dying frogs. It seems to be in accordance with some natural law that rhythmic phenomena tend to become modified when the structures concerned have their vitality lowered in any way. This conception appears to be confirmed by Steiner's observations on pelagic acalephæ. The rhythmic contractions of the calyx fall into periodic groups, separated by pauses, when these animals are kept in water which has not been renewed sufficiently often.

Appearances analogous to those seen in Cheyne-Stokes' respiration are observed in various conditions. Changes in the rhythm lead in many cases to more or less periodicity of groups of pulsations, and in certain circumstances, more especially perhaps when the heart has undergone

degenerative changes, the pulse pressure manifests a marked tendency to periods of elevation and depression. The Traube-Hering curves seen on physiological investigation are probably in every way analogous to this symptom. Reasons were previously adduced by me for the belief, that in certain states of the nervous system, there are periodic fluctuations of consciousness which may be regarded as similar to the periodic changes of respiration.

An essential of Cheyne-Stokes' respiration is a periodic variation in the functional activity of the automatic centre for respiration. Whether the periodic variation is simply dependent upon the loss of the influence of the higher regulating centres, or whether it is also at the same time the result of diminished vitality in the respiratory centre itself, is not at present in our power to decide.

There are two modifications of the respiratory process which sometimes form characteristic features of cardiac disease.

*Yawning.*—In some instances of cardiac dilatation there have been in my experience great tendencies towards yawning, tendencies often absolutely uncontrollable by any effort of will on the part of the patient.

*Hiccough.*—This is an occasional and sometimes troublesome accompaniment of cardiac disturbances.

*Cough.*—This is frequent in cardiac disease, and its causation is manifold. It may result from slight bronchial catarrh, forming one end of a series of phenomena, or from pronounced œdema of the lungs, in consequence of long-standing venous stasis, furnishing the other. It may sometimes take its origin in irritation produced by transudation into a pleura. The cough is often attended by characteristic expectoration. In slight bronchial affections there may be merely a small amount of muco-purulent sputum, while in that emanating from a water-logged lung the sputum is very copious, consisting largely of serous fluid, in many cases deeply stained with blood. It is almost invariably very frothy in its characters.

*Hæmoptysis.*—Amongst the pulmonary symptoms of cardiac disease hæmoptysis must be ranked as one of the most striking. It is found more particularly amongst patients suffering from mitral disease. Germain Sée has called particular attention to

this symptom as one of the earliest manifestations of pure mitral obstruction, and Peter has pointed out that hæmoptysis may be one of the earliest, in fact even the first symptom of mitral incompetence. The explanation of some of these instances of hæmoptysis is not quite clear. Sée will not allow that the cause is to be sought in an increase of cardiac energy due to hypertrophy, and he is inclined to regard the hæmorrhage as due to changes in the walls of the vessels, such as the varicosity described by Buhl, and the degenerative changes so well known. He will not admit that any hypertrophy exists during the early stages of mitral disease, but it seems to me that his reasoning is faulty in this respect. It is probable that the thickening of the vascular walls, originally described by Wilson Fox, serves as a conservative influence, tending to lessen the liability to hæmorrhage.

In many instances in addition to the characteristic expectoration there may be suggestive physical signs, such as slight comparative dulness on percussion over a limited area of the lung, a harsh character of the breath sound, even tending towards a bronchial type, but apt to be obscured by various moist accompaniments and attended by an increase of vocal resonance.

It occasionally happens that in the course of cardiac disease there may be signs of consolidation connected with one or other apex of the lungs. In such a case as this there will of necessity arise considerable discussion as to the origin of this condition. In the presence of increased vocal fremitus, dulness on percussion, bronchial breathing, and increased vocal resonance, with or without auscultatory accompaniments, there may be a suspicion of some tubercular affection. As is well known, phthisis pulmonalis is very seldom associated with mitral disease, and on examination of the sputum it is commonly found that there are no tubercular bacilli in it. On post-mortem examination of such cases it is usually found that there are simply evidences of long-continued venous stasis with brown induration, and sometimes a few of the results of capillary rupture which have not yet been entirely removed.

SYMPTOMS CONNECTED WITH THE INTEGUMENTARY SYSTEM.

—In the cutaneous tissues there are frequently manifesta-

tions of changes in the peripheral circulation from which important diagnostic indications may be drawn.

*Colour.*—The integument may be characterised by *pallor*. This is more particularly seen in such cardiac affections as aortic incompetence, in which there is a deficiency of blood in the peripheral circulation. In simple weakness of the heart there is apt to be also a certain amount of paleness, on account of the lack of driving power on the part of the heart. In all such conditions, more particularly when, as in aortic incompetence, there are great oscillations in the blood pressure, there is a tendency to alternate flushing and pallor of any part of the skin; this capillary pulse, which is fully described in another chapter, is of considerable value in diagnosis.

*Cyanosis* may have its origin in any cause which interferes with the oxygenation of the blood, and there may consequently be many factors in its production. It may have its origin, for instance, in any cause preventing the access of air to the blood circulating in the lungs, such as changes in the bronchial tubes, in the lung itself, or in the pleural cavity; it may be produced also by changes within the abdomen, in consequence of which the pulmonary functions are interfered with. With such causes as these we have in this place nothing to do.

Cyanosis is a frequent symptom of cardiac failure, of valvular disease, and of septal defect. It certainly in its most pronounced manifestations is very characteristic of congenital heart disease. A high degree of cyanosis is nevertheless often found towards the termination of life in chronic heart disease, as well as in acute heart failure. A prolonged controversy has existed regarding the causation of the cyanosis of congenital heart disease, and two different explanations of its origin have been supported by numerous observers from the time of their inception until the present day. One theory, originally indicated by Morgagni, is that cyanosis is essentially produced by venous stasis. This view has been supported by Bertin, Louis, Rokitansky, Moreton Stillé, and Peacock, while Laennec also seems inclined towards it. Chevers also gave his adhesion to this explanation with some slight modification. The other, that it depends upon a mixture of arterial and venous blood, is usually but



erroneously attributed to William Hunter, and has been adopted by Corvisart, Gintrac, Paget, Williams, and Hope. Walshe also in his earlier writings ranged himself on the side of this theory, but was afterwards disposed to a middle course. The preponderance of modern opinion is in favour of the view of Morgagni, and it may be said that the explanation of intermixture, as a sole cause, is absolutely untenable. Profound cyanosis is often present under circumstances in which there can be no possible mingling of arterial and venous blood—a fact observed nearly two hundred years ago by Vieussens—while in some instances where arterial and venous blood are allowed to mingle freely, cyanosis is conspicuously absent. In one most interesting observation by Breschet, where the left subclavian artery arose from the pulmonary artery, there was not a trace of cyanosis in the left arm. Cyanosis, therefore, is one of the results of venous stasis from obstruction to the circulation. The venous stasis may be produced by any of the causes which have been previously discussed. It may, for example, be a merely local phenomenon produced by hindrance to venous return in consequence of obstruction, due to causes within a venous channel, or acting upon it from without. It may, on the other hand, be general, having its origin either in a diminution of the forces which propel or attract the blood. It is not denied that when, in addition to some obstacle to the onward flow of the blood, there is also an intermixture of venous and arterial streams, a higher degree of cyanosis may be produced than is possible by means of stasis alone. To take up such a position would be simply to shut the eyes to one of the most common observations connected with congenital heart disease.

The appearances in cyanosis are very characteristic. The tint of the skin is dusky, varying between a slight lividity and a deep purple or violet colour. This is best seen in the parts of the body farthest removed from the centre of the circulation. It is to be observed, for instance, most particularly around the nostrils, in the ears, and in the hands and feet, more especially in the nails. The mucous membranes are all dark coloured, and the superficial veins upon the sur-

face of the body are distended, more particularly upon the face. This may be seen in the mucous membranes, and on examining the retina the veins are found to be turgid and tortuous.

*Jaundice* has already been referred to in the remarks upon digestive symptoms.

*Moisture*.—The amount of perspiration is subject to great differences in circulatory disturbances. In some of the acute diseases affecting the heart it is greatly increased, while many patients with loss of compensation are bedewed with cold sweat. Probably in both these extreme cases there is a paralytic condition of the vaso-constrictor mechanism.

*Swelling*.—It has been seen in a previous section that all the tissues of the body are bathed in lymph which transudes from the blood vessels, but that there is never more of this fluid than suffices to bathe the tissues and to render possible the chemical changes in them. Under abnormal circumstances, however, this condition of matters becomes changed, and an excessive amount of fluid is present in the tissues as well as the serous spaces throughout the body, constituting the condition known as œdema. The accumulation of fluid is more likely to occur in tissues which are lax, and in spaces where a negative pressure forms an attractive force.

The appearances produced by œdema of the subcutaneous tissues vary considerably according to the region of the body, the posture of the patient, and the cause of the œdema. It may, however, be said that there is always an increase in size, a loss of form, and a reduction of elasticity. There is an absolute augmentation in the bulk of the affected part, and this always tends to the production of a circular form. The natural prominences are veiled in consequence of the accumulation, and on pressing the finger upon the tissues invaded by the excessive fluid, a depression is left behind which often remains for a considerable time. The accumulation of fluid in the different serous spaces of the body has also a tendency to produce increase in size and loss of form. Hydrothorax, for example, almost invariably increases the girth of the chest, and produces a bulging of the sides. Hydropericardium causes an increased bulging of the præcordia, and hydroperitoneum

gives rise to a considerable increase in the size of the abdomen.

The influence of gravitation is of considerable importance, but is by no means equal in its effects in the œdema arising from different causes. It is seen in its highest expression in cardiac, and in its least pronounced form in renal œdema.

The amount of fluid bathing the tissues depends upon the balance between transudation and resorption, that is, between afflux and efflux. This may be brought about by several different factors which fall naturally into certain groups. Changes in the blood pressure constitute the most important causes of œdema. These causes may be local or general, but they always involve an alteration of relative arterial and venous pressure. Lower, more than two hundred years ago, showed that œdema of the lower limb might be produced by obstruction in the iliac veins and vena cava, and although doubt was thrown on the fact by Hodgson, it has ever since been known, that, if all the veins returning blood from one part of the body are obstructed, an œdematous condition inevitably ensues, which takes place more rapidly, as Ranvier showed, if there be a change in the nerve influence of the part.

What is true as regards local œdema is applicable to the more general forms, and obstruction to venous return is of all causes of œdema the most powerful. The obstruction to return of venous blood may have its origin in several different conditions. The most important factor is a loss of the aspiratory forces of the heart, and of the negative pressure in the great veins.

It must be remembered that the integrity of the lymphatic circulation is necessary for the proper performance of the functions of resorption, and that when there is any increased pressure in the great veins, there is apt to be an obstruction at the mouth of the thoracic duct as it enters the subclavian vein, which must of necessity interfere with the lymph return.

Changes in the walls of the blood vessels play without doubt an important part in the development of œdema. From the investigations of several observers, more particularly of Reuss, there can be no doubt that capillaries and membranes in different parts of the body are possessed of varying degrees

of permeability, as is proved by the different amounts of the constituents transuded in the various regions of the body. According to Reuss, the transudate of the ventricles of the brain only contains 1.44 per cent. of albumin, while that of the subcutaneous tissues contains 5.79, of the peritoneum 11.14, and of the pleura 18.33 per cent. It must be allowed that other factors may be at work in producing these differences, but that they are possible necessarily involves differences in the permeability of the tissues.

The researches of Heidenhain led him to conclude it to be exceedingly probable that the endothelial cells of the walls of the blood vessels possess some of the properties of secretory cells, and caused him to indicate the likelihood that under altered conditions there may be considerable modifications in these functions.

The permeability of the vessel walls becomes increased by alterations in blood pressure and in blood composition. In long-continued alterations of the relative pressure within the arteries and veins, the structure of the capillary walls undergoes changes which permit an increased amount of fluid to pass out into the tissues, while changes in the relative amount of the constituents of the blood, such as a diminished amount of albumin or a lessened quantity of oxygen, must seriously impair the nutrition and functions of the walls.

Alterations in the composition of the blood play some part in the evolution of the œdematous condition. Hydræmic œdema is the term which has been applied to dropsical accumulations, believed to be produced in this way. Cohnheim and Lichtheim attempted by experiment to ascertain whether such a conception rested on any basis of solid fact. These observers found that on rendering the blood of the dog artificially hydræmic by the injection of large quantities of 0.6 per cent. solution of salt, there was a great increase in the amount of fluid transudation given off into some of the tissues. The result of their observations showed that the œdema produced in this way did not affect the structures usually affected in general dropsy. Cohnheim and Lichtheim concluded from their experiments that œdema produced in this way owed its origin to changes in the walls of the vessels



following upon the altered composition of the blood. It may be added in this connection that Cohnheim has enunciated the view that the subcutaneous œdema in renal affections is due to alterations in the blood vessels of the skin.

In profound anæmia there is a great tendency to œdema, but in this condition, on account of the impaired nutrition of the heart and of the blood vessels, the three different classes of factors which we have seen to be operative in the production of œdema may be at work.

The tendency to œdema produced by alterations in the blood pressure varies according to the conditions of the circulation in the different organs and structures of the body. Lichtheim, for example, has shown that most of the pulmonary veins may be occluded without the production of œdema of the lungs, and this is probably due to the very free anastomosis which takes place between the pulmonary vessels.

It has already been shown that gravitation has much more influence over the circulation in conditions of low blood pressure, and this fact accounts for the early appearance of œdema in the dependent parts of the body under conditions in which the arterial pressure is low and the venous pressure is high.

The production of œdema in the form of anasarca of the connective tissues or effusions into the serous sacs presents many problems, which have not as yet received satisfactory solution. Œdema consists in the accumulation of fluid which has transuded from the vessels into the spaces of the connective tissue, or into the serous sacs, and it must depend upon some disturbance of the equilibrium between the production and absorption of lymph.

The formation of lymph depends upon two distinct factors: (1) the blood pressure in the capillaries, and (2) the permeability of the walls of the capillaries. The process of lymph formation, therefore, is somewhat similar to that of filtration, inasmuch as an increase of pressure or diminution of resistance will increase the fluid passing through. The result of all the experiments made on the flow of lymph from the lymphatics of the limbs proves that great changes in the pressure of the capillaries, that is to say, in the filtration pressure, give rise

to very slight effects on the lymph flow—very slight indeed as compared with those which would be produced by equal changes in the vessels of the abdominal viscera. The conclusion is obvious that the capillaries of the limbs present great resistance to filtration.

The filtrate through a membrane exhibiting considerable resistance contains a smaller proportion of proteids than the original fluid. Starling suggests that whenever a proteid solution is filtered through a membrane showing considerable resistance to proteids, a certain amount of energy is used up in the process, the hydrostatic pressure used for the filtration being converted into an increased osmotic pressure of the concentrated fluid. In such a case the fluid within the cell or vessel will attract the more watery fluid outside with a force equal to the difference in the osmotic pressure of the two factors, while the force giving rise to the process of filtration or transudation is to be estimated by the excess of the hydrostatic pressure inside the cell or vessel over this difference in the osmotic pressure.

Starling ingeniously applies these principles to the conditions within the body. When the blood goes from the arterioles into the veins by means of the capillaries it passes from a higher to a lower pressure. The capillaries may therefore be regarded as falling into two regions. In the arterial capillaries, the plasma is under a pressure exceeding the osmotic pressure of the proteids of the plasma, from which therefore the transudation takes place of a fluid poorer in proteids than the plasma. This fluid bathes the outside of the venous capillaries, in which, however, the blood pressure is below that corresponding to the osmotic pressure of the proteids of the plasma; and here, therefore, the force tending to the absorption of the water and salts,—the osmotic pressure of the plasma, is greater than the force giving rise to transudation,—the hydrostatic pressure of the blood in the vessels. This absorption can only influence water and salts, as there is no means of absorption of proteids outside the vessel walls. The result is the continual transudation of a fluid relatively poor in proteids from the arterial capillaries, and a reabsorption of its water and salts by the venous

capillaries. Hence the fluid remaining in the tissue spaces must contain a higher percentage of proteids than when it left the arterial capillaries. This fluid, in so far as it is not made use of by the tissue elements, must be collected by the lymphatics and returned to the vascular system by the thoracic or right lymphatic duct. When the lymphatic channels are blocked or obliterated, failure of absorption follows on account of the inability of the blood vessels to absorb fluids containing more than a certain percentage of proteids. These researches do not support the views of Heidenhain.

As has already been shown, the effect of heart failure is to produce diminution rather than increase of capillary pressure, which is followed by increased absorption from connective tissue spaces, and therefore a smaller amount of blood in them. One of Cohnheim's experiments throws some light upon this subject. Ligature of one femoral vein in a dog does not produce œdema, but Cohnheim found that by bleeding a dog several times during one or two weeks before ligature, it was followed by œdema of the leg. If, on the other hand, hydræmia is produced just before ligature, by bleeding and injecting saline solution, no œdema follows. Cohnheim therefore concludes that long-continued hydræmia interferes with the vessel wall, rendering it more permeable, and that the rise of capillary pressure, ineffective under ordinary circumstances, is able to increase the transudation of lymph and cause distension of the tissue spaces. There is no plethora in heart disease, but there is hydræmia, and the nutrition of the wall is further affected by the failure of the circulation and the consequent disturbance of metabolism.

The greatest difficulty in assuming such a view to account for the œdema of heart disease lies in the fact that the fluid in œdema contains only from 0·3 to 0·5 per cent. of proteids, that is, less than that found in the lymph from the limbs of normal animals; but it has to be remembered that the original blood plasma is in itself poorer in proteids in cases of heart disease than under ordinary circumstances, and the chief factor tending to increase the concentration,—absorption by the blood vessels, is greatly interfered with. The effect of the slow blood current is undoubtedly to approximate the venous

and arterial pressures, so that if the hydrostatic pressure in the arteries can cause abnormal transudation, there will from increased venous pressure be an abnormal transudation from the veins also. Absorption by the blood vessels will then only be possible when the capillary pressure is reduced by elevating the part.

**SYMPTOMS CONNECTED WITH THE URINARY SYSTEM.**—It is a well-known fact, as Saundby says, that the effect of heart disease is to approximate the mammal to the reptile as regards the urinary system. All oxidation processes are diminished, and therefore the formation of uric acid and its congeners is increased at the expense of urea. As a consequence of this tendency, the kidney always suffers to a greater or lesser extent whenever backward pressure affects it. As a result of these alterations, there is considerable tendency to development of chronic interstitial changes in the kidney.

The effect of heart disease in producing renal cirrhosis has been emphasised by Dickinson, and there can be no doubt that in long-continued circulatory interference with the nutrition processes, there is frequently a development of the latter condition. It is, however, open to question whether there is not a deeper underlying relationship in some such common cause as arterio-sclerosis. My own opinion runs strongly in this direction, and somewhat similar views are expressed by Hamilton.

But this is only one aspect in the relations between cardiac disease and renal disturbance, as a more direct implication of the kidney results from venous stasis. In long-continued backward pressure resulting from any disturbance of the circulation, cyanotic induration is developed.

The characteristic morbid appearances in cyanotic induration are, an increase in the size and weight of the kidney, which becomes harder in its consistence, but has, however, little tendency towards adherent capsule. On stripping off the capsule, the surface is usually smooth and of a deep purplish-red tint, while on section there is enlargement both of the medulla and cortex, and the cut surface is of a deep purple colour until, after exposure to the oxygen of the air, it



becomes brighter in hue. The Malpighian bodies are large. On microscopic examination profound hyperæmia of the capillaries forms the peculiar feature of cyanotic induration, and this is more especially the case towards the surface. There is, not infrequently, a considerable infiltration of the Malpighian bodies with small round cells. The epithelium of the tubes is swollen, but no great desquamation can be seen. Hæmorrhage into the tubes is not infrequent. It is often said that some cirrhosis is present. When this, however, is the case, the cirrhosis is due to antecedent or concomitant causes and not to the venous stasis.

The urinary symptoms of cardiac disease are tolerably uniform. The amount of secretion is invariably diminished, its specific gravity raised, and its colour deepened. It is very often turbid on standing a few hours, and it very commonly throws down copious deposits of urates. Albuminuria is frequent, and sometimes the amount of albumin is large. From the condition of the kidney already referred to, it is probable that the escape of albumin is permitted both by the Malpighian tufts and by the intertubular capillaries. The suggestion may be thrown out that, just as in the production of œdema some antecedent alteration in the vessels has taken place, so in albuminuria there is not merely obstruction of the venous return, but some deterioration in the walls of the vessels. Blood often appears in the urine in chronic cardiac affections as the result of extravasations from rupture of the capillaries. Tube casts are often found, and are usually hyaline or granular, but blood casts frequently appear.

#### SYMPTOMS CONNECTED WITH THE REPRODUCTIVE SYSTEM.—

As consequences of circulatory disturbances, there are often interferences with the functions of the reproductive organs. Some of these are merely results of alterations in the blood pressure, and are therefore symptomatic; others speak for a profound modification of the system at large.

Amenorrhœa is extremely common in chronic heart disease, especially in mitral lesions, and the more so when there is a tendency to anæmia. It may be regarded as a natural process of compensation by diminution of loss. Menorrhagia is often found when there is deep venous

stasis, and undoubtedly results from the hyperæmic state of the pelvic organs. Leucorrhœa like menorrhagia is a consequence of backward pressure, and is simply an expression of a catarrhal state of the mucous membrane.

Pregnancy, parturition, and lactation might well be expected to have some relation to circulatory disorders, and there is indeed a reciprocal influence between them. Not only do grave disturbances of the circulation impair fertility and produce abortion, but they render parturition dangerous, and lactation difficult; while, from the other point of view, the incidence of these generative phenomena are attended by serious risks, and frequently end in disaster. These mutual relations between the circulatory and the reproductive functions have been carefully studied by many authors, more especially by Macdonald and Peter.

SYMPTOMS CONNECTED WITH THE NERVOUS SYSTEM.—The existence of painful subjective sensations in circulatory disturbances has long been known. In the first decade of last century such symptoms were observed by Morgagni, and about fifty years later described by him. Since that time they have greatly occupied the attention of the physician, but the scientific investigation of the subject has, nevertheless, made but little progress until recently.

The sensory disturbances are of different kinds: uneasiness, tightness, weight, and oppression are varying degrees of subjective sensation which culminate in a feeling of pain. One and all of these sensory phenomena may be accompanied by sensations of sinking, of fainting, of dying; or attended by feelings of disturbed movement within the chest, of troubles affecting the respiratory processes, and of disorders connected with the cerebral functions.

The pain in cardiac affections varies infinitely in degree. Between the slight uneasiness of an evanescent heart strain and the profound anguish of fatal angina pectoris there seems to be a wide gulf; there is, nevertheless, no absolute distinction between them—the difference, as elsewhere stated by me, is not in kind, but in degree. The milder examples of pain are linked with the more severe by an unbroken series of intermediate forms, and it often happens that in the same individual a feeling not

so much of definite aching as of vague discomfort passes into such severe pain as to produce intolerable agony.

Cardiac pain is usually exaggerated by exertion, and sometimes is only experienced after muscular effort, more especially in ascending a hill or a stair. All kinds of excitement tend to induce it, and a full meal is liable to be followed by increase of pain. Needless to say, when several of these factors act simultaneously, the effect is more marked.

The pain in cardiac disease is not, for the most part, felt by the patient in the heart, but is chiefly referred to the surface of the body. From the time of Harvey it has been known that the heart is by no means sensitive to painful impressions. It is no doubt true that certain sensations are referred to the heart itself—feelings of disturbed movement are often present; pain, however, is always, on careful investigation, found to be superficial.

The usual situations of pain are in front of or behind the chest, corresponding to the distribution of the spinal nerves. The most common nerves affected are those arising from the eighth cervical and the upper six dorsal segments of the cord, but pain is also felt over the areas connected with the cord as high as the third cervical and as far down as the lower dorsal nerves. In some cases there may be pain over the spines of the vertebræ corresponding to the areas affected, particularly in the lower cervical and upper dorsal regions. The pain, further, is felt in the areas of the upper limbs innervated from the spinal segments implicated.

Pain resulting from affections of the heart and aorta is sometimes confined to one half of the thorax, sometimes it involves both sides. When unilateral it is far more commonly situated over the left than the right side; when bilateral it is almost always symmetrical.

Tenderness is almost invariably present when there is pain, and this increased sensitiveness is often found when there are no subjective sensations. The best method of testing this exalted sensibility is by gentle pressure with a fine point; over areas not affected, the stimulus is only felt as a touch, but when the hyperalgesic region is entered there is a painful sensation.

Another very simple and most effective method of testing the sensibility of the skin consists in gently seizing part of the area suspected of exalted sensitiveness between the thumb and first finger of one hand, while another part, exactly corresponding on the opposite side of the body, is similarly grasped by the other hand. By slightly and equally squeezing the two parts so seized, it is easy to determine which is the more tender to pressure. In some cases this gentle stimulus is sufficient to excite a paroxysm of pain, as Mackenzie has shown.

The tenderness was thought by Peter to be situated in the heart itself; this, however, is not the case. It is easy to determine that it has its seat in the superficial textures of the thorax. Not only the integumentary structures, but the underlying muscles also, are tender; the pectorals, the sternomastoid, and the trapezius of one or both sides may be sensitive to gentle pressure. The increased sensitiveness of the surface of the body in painful cardiac affections did not entirely escape the acute observation of Laennec, but he gives this aspect of the subject very scant notice. Although the terms employed by Laycock are not free from ambiguity, he seems to have grasped the fact of hypersensitiveness of the skin in cardiac pain; the great value, however, of his remarks on the subject lies more in their suggestiveness than in their fulness.

The explanation of the pain of cardiac disease by a study of the relations of the cardiac nerves to the spinal cord was suggested by Lussana, who traced out the connections of the cardiac nerves, the spinal segments, and the brachial plexus; it was further advanced by Sturge; and the segmental distribution of such pain arising from visceral disturbance was clearly brought forward by Ross. A great step was taken by the brilliant and original researches of Mackenzie into the presence of exalted sensibility, as well as of pain, in these visceral affections. His painstaking observations on the exact areas of subjective and objective sensory changes constitute a most important advance, and mark the commencement of a new epoch. The beautiful and elaborate series of observations made by Head on this subject have still further elucidated many of the difficult problems connected with the sensory symptoms of visceral disease.



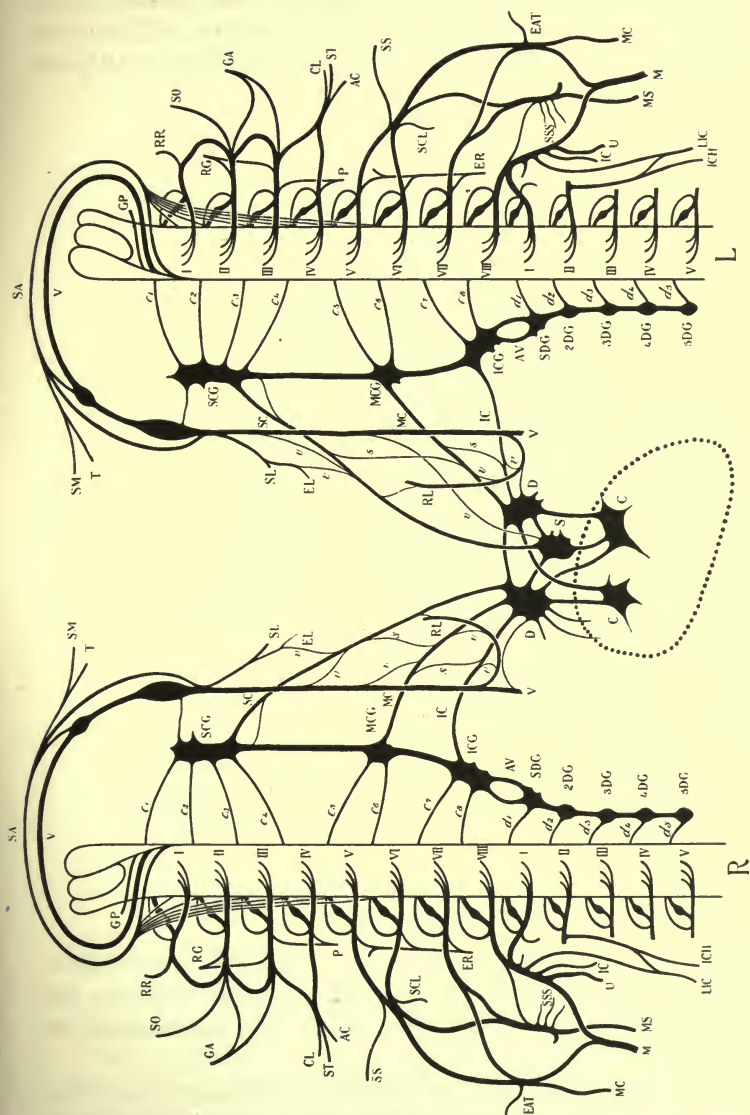


FIG. 104.—Diagram of the connections of the cardiac nerves. For description see text, p. 24.

The connections of the cardiac nerves have been described at length in a previous section (p. 22) and need not be repeated, but the accompanying diagram, Fig. 104, will serve to render the subject clear at a glance, and the Figures 105 and

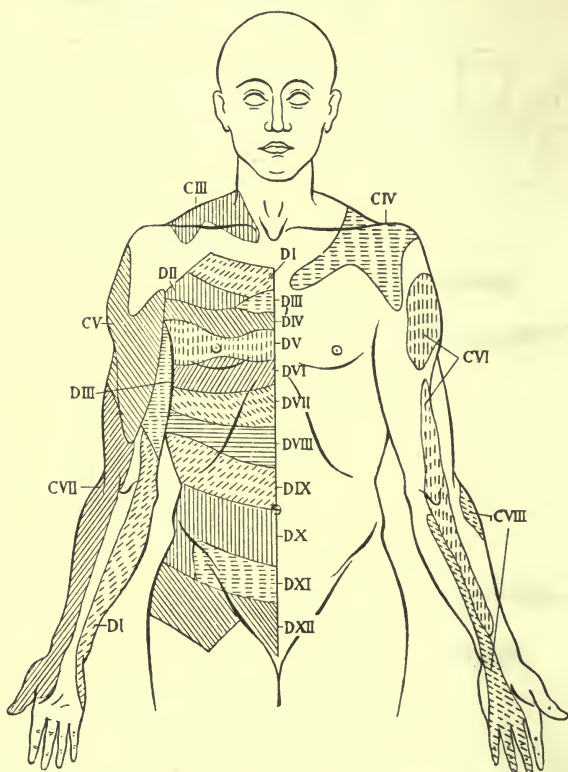


FIG. 105.—Cutaneous distribution of the sensory nerves.

106, constructed from Head's tracing, show the sensory distribution of their connections. In these figures the letters and numerals indicate the segments of the cord with which the nerves are connected.

It may be regarded as established beyond doubt that afferent impulses travel upwards by means of the sympathetic as well as the pneumogastric nerves. The distribution of the sensory disturbances in disease, involving areas which can

only be reached through the two channels, is in itself an ample proof of this fact. Of the two paths, that furnished by the sympathetic system is most frequently implicated, and this more especially in its lower cardiac attachments. The inferior

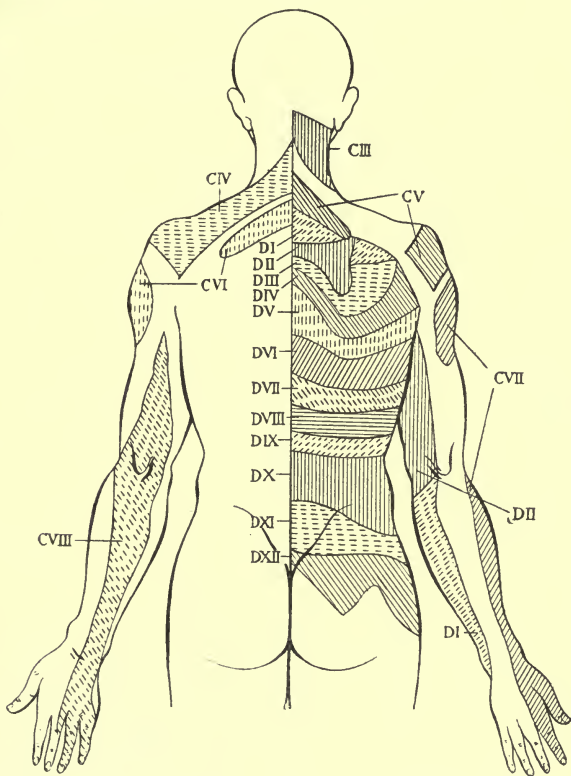


FIG. 106.—Cutaneous distribution of the sensory nerves.

cardiac nerve, taking its origin from the inferior cervical and the first dorsal ganglia, is the afferent channel by which impulses seem most commonly to reach the centres.

The sympathetic ganglia are connected with the spinal nerves by two different kinds of communicating branches, the gray and the white, which have been thoroughly investigated by Gaskell. The superior cervical ganglion is connected with the first, second, third, and fourth cervical nerves; the middle

with the fifth and sixth; the inferior with the seventh and eighth. According to Gaskell, the heart receives its visceral nerve supply mainly from the second dorsal pair.

The accompanying diagram, Fig. 104, gives the various connections of the cardiac nerves with the spinal segments and the superficial nerves.

The sensory path enters the cord by the posterior root and passes upwards to the brain. It is almost certain that its course is interrupted at least three, perhaps even four, times—in the sympathetic ganglion, in the posterior root ganglion, and in the gray matter of the cord, probably also in the optic thalamus. An impression upon the end-organ always produces an influence upon the centre, with which it is in direct connection. We are ignorant of the nature of the effect that occurs in consequence of this influence, but that some molecular change takes place cannot for a moment be doubted, for, as Virchow says, “jede Function ist an mechanische Veränderungen der Substanz geknüpft.” Sometimes the effect of the impression is sufficiently powerful to reach the higher centres, where it can be perceived; at other times it does not travel beyond the proximate centre. That the effect of a stimulus is not always conducted to the brain, is proved by the existence of hypersensitiveness without pain, which has been specially referred to by Nothnagel and Mackenzie.

The heart, like most of the viscera, is but slightly sensitive. A sensory stimulus is carried to the cord in the region from which the sensory nerves have their origin, and in that segment comes into relation with the nerves for painful sensation connected with the segment. “But,” as Head has so well put it, “the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus, by what might be called a psychological error of judgment, the diffusion area is accepted by consciousness, and the pain is referred on to the surface of the body instead of on to the organ actually affected.”

The explanation of tenderness in association with pain which is given by Head is worthy of careful consideration, and merits general acceptance. Impulses passing to the cord from a diseased viscus produce a disturbance in the segment to which



they pass, and induce a condition of unstable equilibrium, so that any stimulus applied to the area connected by sensory nerves with the segment will be more powerful in its effects and will give rise to exaggerated sensations.

The greater frequency of pain in the left half of the chest can only be due to the fact that the left ventricle and its attachments are connected mainly with the left coronary and left deep cardiac plexuses, which, along with the entire superficial cardiac plexus, are connected with the left nerve centres. The left ventricle and aorta are, on account of the higher pressure to which they are subjected, much more liable to degenerative changes than the right ventricle and pulmonary artery. And as the left ventricle has no means of relief from strain, such as is provided for the right by the safety-valve functions of the tricuspid valve, long-continued excess of pressure acts more seriously upon it.

It is scarcely necessary to mention that pain in the head has been from comparatively early times recognised as accompanying heart affections. No exact observations, however, seem to have been undertaken to ascertain the relations of this pain until the elaborate investigations of Head made their appearance. He gives good reasons for believing that certain areas of the forehead and scalp are associated with some of the thoracic regions. The naso-frontal area, for example, is related to the distribution of the third and fourth cervical nerves; the mid-orbital, with the second and third dorsal; and the fronto-temporal, with the fifth and sixth dorsal nerves.

Upon all the aspects of this subject, in so far as they have been touched upon, modern writers are in substantial agreement, but on inquiring into the origin of the impulses travelling towards the sentient centres, a great diversity of views is unfolded. It would be out of place in this sketch of subjective symptoms to attempt a criticism of the varying opinions which have been held regarding their immediate causation; it is of more importance to ascertain whether the sensory disturbances may be classed in etiological groups. That they are produced by many diverse influences cannot for a moment be doubted, and by a process of analysis they may be arranged in distinct classes.

That pain having its origin in the sensory nerves of the

heart may occur in conditions of anæmia must be admitted, and instances of this kind may be considered analogous to forms of neuralgia occurring in cases where the blood is deficient in corpuscles or hæmoglobin. The exact nature of the change in the nervous structures in such cases is matter for speculation. It may be merely an irritable weakness, but, as in anæmia there are often profound structural alterations in the nervous system, there may be definite lesions in some part of the complex mechanism.

Toxic influences constitute a frequent source of cardiac pain. Chief amongst the agents belonging to this class are alcohol, tea, and tobacco. There is no general rule as to the relative potency of these substances; to some persons one of them may be a powerful poison, while the others are comparatively harmless; it may be accepted, however, as the result of experience, that tobacco gives rise to more pronounced and more persistent sensory effects than the others. In the greater proportion of such toxic cases absolute recovery takes place, and the production of the sensory symptoms is, therefore, probably due to a chemical combination between the poison and the protoplasm of the nervous structures—not to any degenerative change in the latter. It is only right, however, to add that in many of these cases undoubted lesions of the heart are present.

Toxic influences may arise from faulty metabolism within the body. Lithæmia and glycæmia are the best examples of such auto-intoxication affecting the nervous connections of the heart, as they have not only been recognised for a considerable time, but are of comparatively frequent occurrence. The mode of operation is most likely analogous to that of poisons introduced from without.

Direct pressure on some of the cardiac nerves by sclerosed coronary arteries was thought by Home to be the cause of pain in the classical instance of John Hunter; analogous pressure was seen by Heine to be the apparent cause of certain of the symptoms of angina pectoris; and pressure on the phrenic was found by Haddon in a pronounced case of angina pectoris. In this last case, however, there were aortic lesions, and the implication of the phrenic nerve may probably have been an accidental concomitant.

Neuritis of some of the cardiac nerves and plexuses was found by Lancereaux in association with aortic disease and coronary sclerosis in angina pectoris, and a similar observation was made by Peter in a case in which, however, coronary aortic, and pericardial lesions were found after death.

Organic disease of the heart is present in a large proportion of instances of cardiac pain. Simple cases of heart strain, and dilatation of the heart from almost every cause, furnish examples of slight changes accompanied by sensory symptoms. Degeneration of the muscular wall, and lesions of the aortic orifice or its cusps, as well as changes in the walls of the aorta, are likewise associated in a large number of cases with such painful sensations; but above all, arteritis deformans of the coronary vessels, linked, as is almost invariably the case, with consecutive changes in the myocardium, is the underlying lesion in the graver painful affections connected with the heart.

Pain is of very frequent occurrence. Attempts have been made to ascertain the relative proportion of cases in which it is present, and Nothnagel, in a recent investigation of 483 cases of valvular disease under treatment in his wards during seven years, gives the following results:—

Nature of Disease.	Sensory Symptoms.		Percentages.	
	Present.	Absent.	Present.	Absent.
Aortic incompetence . . . . .	69	45	60	40
Aortic incompetence and obstruction . . . . .	15	8	68	32
Aortic obstruction . . . . .	2	3	40	60
Aortic and mitral incompetence . . . . .	3	14	18	82
Mitral incompetence . . . . .	14	169	8	92
Mitral obstruction . . . . .	4	18	18	82
Mitral obstruction and incompetence . . . . .	20	98	17	83

The contrast between the aortic and mitral cases, as regards the incidence of pain, is very striking.

These organic cardiac lesions have afforded a fertile field for controversy in attempting to explain well-known facts, and the difficulties which surround the subject on every side only seem to increase on closer examination. It is not possible to analyse all the different views which have been brought

forward to explain cardiac pain, but a rapid glance at certain of the doctrines which have been advanced may be helpful. The pain was regarded by Heberden and Latham as produced by spasm of the heart itself. It was, on the other hand, regarded as an expression of failure of the heart by Parry and Stokes, and this has been well expressed in recent times by Lauder Brunton, when he says that cardiac pain is generally due to weakness of the heart in proportion to the resistance which it has to overcome. The sensory symptoms have further been attributed to ischæmia of the cardiac muscle. This view was originally propounded by Burns, whose words, with their quaint punctuation, are well worthy of being quoted:—

“In health, when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action, the heart and every other part has its power augmented. If, however, we call into vigorous action, a limb, round which we have with a moderate degree of tightness applied a ligature, we find that then the member can only support its action for a very short time; for now its supply of energy and its expenditure, do not balance each other; consequently, it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence. A heart, the coronary vessels of which are cartilaginous or ossified, is in nearly a similar condition; it can, like the limb, be girt with a moderately tight ligature, discharge its functions so long as its action is moderate and equal. Increase however the action of the whole body, and along with the rest, that of the heart, and you will soon see exemplified, the truth of what has been said; with this difference, that as there is no interruption to the action of the cardial nerves, the heart will be able to hold out a little longer than the limb.”

This theory, espoused with various modifications by many writers, has been revived by Potain, who has applied to it the theory of Charcot termed “*claudication intermittente*.” It is exemplified by the transient paraplegia which follows exertion in thrombosis of the iliac arteries or abdominal aorta. The collateral circulation is adequate when the limbs are not in active use, but when they are exerted a relative ischæmia



causes temporary paralysis. Sée accepts ischæmia as a valid cause, and it is held by Huchard that a spasm of the coronary vessels may cause the pain.

The sensory disturbances were regarded by Laennec as neuralgic in character, and this view has been adopted with more or less modification by many authors, and especially by Anstie. Romberg regarded them as due to hyperæsthesia of the cardiac plexus. That the cardiac pain may have its origin in, or at least be connected with, neuritis, has been placed beyond dispute by the observations of Lancereaux and Peter.

Connected with the idea of increased peripheral resistance, Cahen enunciated some vague speculations regarding the possible dependence of painful sensory symptoms upon some vasomotor influence. Landois attempted to place the subject on a philosophic basis by classifying the various possible causes of painful affections of the heart, and in his contribution he specially mentioned pain arising through vasomotor influence (produced by a spasm of the arterioles), and pain taking its origin in reflex impulses.

Nothnagel, following somewhat in his footsteps, further expanded the idea of vasomotor pain, and described anginous cases without cardiac lesions, in which numbness of the extremities, induced by exposure to cold, was followed by præcordial pain, palpitation, and faintness, accompanied by a pulse of small size. Eichwald advanced the opinion that they might be produced by the efforts of a weak heart to overcome peripheral resistance by excessive activity.

The careful clinical observations and brilliant therapeutic suggestions of Lauder Brunton, so well known as to require no description, gave powerful support to the belief that in at least a proportion of cases the pain of angina pectoris had its origin in a vasomotor spasm. The pulse changes, however, described by many observers, are by no means constant, as has recently been clearly shown by Morison; and relief does not always follow the use of drugs which dilate the vessels, a circumstance originally noticed by Fagge. These undeniable facts are in themselves enough to demonstrate that vasomotor influences are not a valid explanation in all cases.

The more modern views bring us face to face with the

probability that a large number of different factors are at work in producing irritative impulses, which travel by the afferent nerves to the centres, and there produce an explosion of nerve energy which is referred to those peripheral tracts corresponding to the central segments involved. This conception probably contains the truth. Malnutrition of the sensory endings, as in general anæmia or local ischæmia; toxic influences, whether organic or chemical; direct irritation, as in the hyperæmia of pericarditis; interference by means of structural changes—all produce stimuli which either travel directly in a centripetal direction, or give rise to a condition of irritability in which, when an additional strain is undergone (if the heart is struggling to overcome a resistance to which it is inadequate), afferent impulses will be carried to the nerve centres.

Very masterly criticisms of the theories regarding the pain of angina pectoris are furnished by Gairdner, Huchard, and Osler. No one can read their well-weighed conclusions without being convinced that the origin of cardiac pain is certainly manifold. This is also urged by Douglas Powell and by Grainger Stewart. Balfour, in his latest work, states that, like other neuralgiæ, angina has its origin in a lowering of the functions of the affected nerves, usually from prolonged malnutrition, which is sometimes brought to a climax by some positive cause of ischæmia such as vascular spasm, or more rarely by pressure on part of the plexus.

Subjective sensations arising from disturbances of the energy of the heart are frequently complained of. Many of these sensations are, however, not connected directly with any affection of the circulation, and take their origin in some reflex influences acting through the nervous system. Of such a class is the throbbing which is produced by emotional excitement and digestive disorders. Such symptoms naturally fall into line with the painful sensations having their origin in analogous causes.

The sensations of motor disturbance experienced by patients present a somewhat variable group of symptoms, and it must be observed that they are very commonly associated with feelings of pain, oppression, and constriction. The sensa-

tion of throbbing commonly known as palpitation consists in greater frequency and increased energy of pulsation. It may be excited by exertion or excitement, and it has an objective as well as a subjective aspect. In most instances it is only seen from time to time when the special exciting cause has called it into being, but it is occasionally present persistently or for long periods. The sensation of fluttering is often complained of in conditions of cardiac failure, and it has been known for long under the name of tremor cordis. A consciousness of irregular pulsation, "a strong beat, then five or six very small ones all in a rabble," as graphically depicted by a patient whose case is given by Balfour, constitutes delirium cordis. A feeling of stopping or arrest of the contractions of the heart is also frequently experienced by patients suffering from failure of the muscular walls. The combination of these different sensations may be present in the same individual. The patient may experience a feeling as if the heart's action had entirely ceased, and this sensation may be followed by violent throbbing, which may in turn pass into the condition of fluttering or trembling. These different feelings of perverted movement are most common when there is serious disorganisation of the myocardium. All these symptoms may be quite independent of any organic cardiac lesion. They may be produced entirely by nervous influences; on the other hand, they may be the result of serious organic disease of the heart. It is quite obvious that in this latter class there are two factors at work. There is, in the first place, the disturbance of the movements of the heart, but, as such interferences with the rhythm and energy of the heart are often present without any consciousness of their existence on the part of the patient, it is clear that there must be, at the same time, some other condition whereby their presence becomes known. This can only be by an increased sensitiveness of the cardiac nerves, and the co-existence of such symptoms along with painful sensations is, therefore, easy of explanation.

The feeling of faintness is a sensory symptom often associated with disturbances of movement, such as have just been described. It takes its origin, no doubt in most instances, in

a failure of the circulation in the nerve centres, such as is present in its fullest development during syncope.

Giddiness is sometimes found in disturbances of the circulation. It is a special sensory symptom essentially dependent upon a lack of the normal relationship existing between the afferent impulses and motor responses. It may, therefore, be produced by changes in the centripetal fibres, the receptive centre, the emissive centre, or the centrifugal fibres. When dependent upon circulatory disturbances, vertigo must be due to changes in the centre brought about by failure of the circulation. Vertigo is, probably, not nearly so often found in the absence of aural disease as used to be supposed; it is, nevertheless, by no means uncommon to find patients presenting well-marked symptoms of vertigo, in whom careful examination reveals some ear defect, in association with which circulatory disturbance has produced a troublesome giddiness.

The special senses are apt to be disturbed when there is any serious alteration in the blood supply to the centres. Such symptoms may be present in connection with any and all of the special senses, but sight and hearing are those most commonly interfered with.

There may be simply dimness of vision, or there may be various visual hallucinations, such as flashes of light, floating specks, or quivering lines, all of which may be dependent upon a loss of the blood supply to the visual centre. There may also be, on account of some local disturbance of the circulation involving the optic tract or nerve, such symptoms as hemiopia, or unilateral amblyopia, or there may even be scotoma of one eye. It need scarcely be added that on examination of the eye with the ophthalmoscope some interesting symptoms may be seen, such as the exaggerated arterial pulse of aortic incompetence, the presence of a retinal embolism, or other local circulatory lesion.

The sense of hearing may undergo analogous disturbances. The hearing may simply be imperfect, or there may be ringing in the ears or some other subjective disturbance. One symptom associated with the sense of hearing, which is present in a considerable number of cases of heart disease, is the feeling of throbbing in the ears attended by a distinct rushing



sound when the blood pressure undergoes great variations, such as is the case in aortic incompetence.

Various sensations are referred to the head. Headache, as has previously been mentioned, is often associated with heart disease. The most common way in which it manifests itself is by a dull aching widely distributed over the head; but, as was shown, there is often headache accurately localised in one spot, and often associated with pain connected with the chest.

There is often a feeling of fulness in the head, not uncommonly expressed as a feeling as if it would burst, and the opposite feeling of emptiness is probably just as common.

It is extremely difficult to give a rational explanation of these morbid sensations connected with the head. We as yet know but little of the structures in which such sensations are seated, and even less of the processes by which they are brought about. One point must never be forgotten, that two sensations absolutely diverse in character may be apparently produced by exactly the same conditions. The sense of fulness or of pressure, for example, and the sense of emptiness may take their origin in absolutely the same state of the circulation.

Sleep often undergoes troublesome changes. It is extremely common to find, in various conditions where the circulation is deficient, that the patient complains of constant sleepiness. Such a symptom is often found in elderly persons with cardiac weakness.

Sleeplessness is a much more distressing symptom and one which is, unfortunately, more common than the soporose tendency. It may show itself as simple inability to fall asleep, or as troubled sleep, interfered with by alarming dreams.

The soporose and the sleepless tendencies may owe their origin to similar, if not identical, causes. We know that sleep is dependent upon the healthy fatigue of the nerve cells, and the only explanation which can be offered of its perversions lies in the assumption that some alteration has taken place in the normal relationship existing between the nerve cells and their blood supply, whereby they are brought into a condition of irritable weakness.

The higher functions of the brain often undergo great alterations when the circulation is disturbed. There may be

a complete arrest of these higher functions, producing more or less complete unconsciousness, or the cerebral processes may be perverted and delirium be present. Instead of such complete changes in the functions of the brain, there may be other and less pronounced effects; such as a deficiency in the power of reasoning and judging, a loss of the faculty of memory, or some alteration, in a more restricted way, of the intellectual functions of the brain. All these varied effects may be the simple result of a diminished supply of blood to the brain, in which the general or local symptoms have their origin; but they are more common in patients who have at once some structural alteration in the nerve centres, and derangement of the circulation.

## CHAPTER V.

### THERAPEUTICAL.

ON account of the complex relations between the circulation and every other system, the treatment of its disturbances involves reference to all the functions of the body. Circulatory affections, therefore, must be managed with regard to the disturbances which they produce, not merely in the special cardiac and vascular mechanisms, but in the functions and structures of the other systems. Only when all the effects produced throughout the body at large have been duly considered, can complete indications for treatment be obtained. In the preceding sections the general pathological considerations have been discussed and the principal groups of symptoms have been explained with reference to their causation. In pointing out the principal indications for treatment, little requires to be done except to attain to some conception of the general principles upon which the therapeutics of circulatory disturbance must be based.

DIFFERENT CLASSES OF DISORDERS REQUIRING TREATMENT.—The circulatory affections which require treatment naturally fall into two great groups—organic and dynamic. The former must be further divided into two classes—acute and chronic. In this section, nevertheless, all these divisions, in so far as is possible, will be considered together; a few general remarks will be made on two or three groups of diseases, but the special details will be left for the particular disease groups which have to be considered afterwards.

INDICATIONS FOR TREATMENT.—The one central fact

around which revolve all the indications for treatment in organic diseases of the heart is that there is a loss of the equilibrium normally existing throughout the circulation. The loss of the normal hydrostatic relations produces a tendency to a diminished amount of blood in the arteries and an excessive amount in the veins, from which there results a liability to the accumulation of fluid within the system. In consequence of these disturbances all the great functions are interfered with. As a result of venous stasis and its effects, there is a catarrhal condition of the alimentary mucous membrane, seriously interfering with the processes of digestion and absorption. From diminution of the air space within the lungs the oxygenation of the blood is imperfect, and this effect leads to consequent morbid changes. The diminution of the renal excretion leads to the retention of waste products which ought to be excreted, while the effects of stasis upon the glomeruli and tubules produce a drain from the blood through albuminuria. The whole nervous system suffers from the disturbed circulatory conditions and reflects its lessened energy to every part of the blood. The entire metabolic functions are in consequence of these different changes directly or indirectly affected; the results upon the blood, and through it upon the heart, are therefore far-reaching. The whole of these disturbances may be said to constitute one large vicious circle.

Arising out of disturbance of the hydrostatic relations of the circulation are several great groups of symptoms which form indications for the employment of therapeutic measures. These have already been somewhat fully discussed, but it may be well to summarise them here from a different point of view.

With regard to the circulatory system but little need be said in this connection; it may, however, from the immediate point of view be stated that the most important indications for treatment are to be derived at least as much from the general symptoms as from the local manifestations of disturbance. Of direct indications connected with the circulation the most important are those furnished by the condition of blood pressure, gauged by palpation of the pulse; by



the rhythm of the pulsation, judged by the regularity and the equality of the pulse wave; by the energy of the heart, estimated by the loudness of the sounds or murmurs; and, lastly, by the effects of circulatory disturbances, evidenced by other systems.

The alimentary system shows the effects of backward pressure by catarrhal conditions of the mucous membranes, revealing themselves by well-marked gastro-intestinal symptoms; the liver is often enlarged, and some degree of jaundice is present; there is frequently hæmatemesis, or melæna; ascites, moreover, is produced.

The hæmopoietic functions become disordered. Some enlargement of the spleen is by no means uncommon, and changes in the blood are so frequent as to constitute the rule. In many instances the amount of hæmoglobin and the number of blood corpuscles is diminished, but, whenever there is profound cyanosis, the hæmoglobin may be increased in amount, through the process of compensation, previously described, and a large augmentation of the corpuscles may be present.

The respiratory system sometimes shows disturbance by cardiac asthma without very obvious physical signs. This is probably in part due to the increased volume and diminished elasticity of the lung tissue; much more commonly, however, there are direct proofs of catarrhal conditions of the bronchial mucous membrane, hyperæmia of the pulmonary parenchyma, or transudation into the pleural cavities. These morbid changes either hinder the access of air to the pulmonary capillaries, or lessen the aerating surface of the lungs, so that there is less possibility of oxygenation of the blood.

The skin and subcutaneous textures become the seat of transudation, and the œdema thus resulting forms an important indication for treatment.

The urinary system shows disturbance by diminution of the secretion, and concentration of the urine. The concentration is compensatory in its effects, yet it is found on analysis that the amount of urinary constituents which it contains is reduced below the proper standard; there is therefore an accumulation of waste products in the system, and consequent evils. Albuminuria is a very common symptom in serious

cardiac disease, and the loss of this important element of the blood plasma forms a grave menace to life.

The nervous system suffers chiefly in its central organs, and sleeplessness, headache, failure of memory, and lack of energy are characteristic indications requiring attention. Such symptoms are, although less frequently, accompanied by attacks of delirium, most commonly at night, and occasionally patients become victims of melancholia, or mania. In many cardiac affections, more particularly of the degenerative type, there is, as has been shown, a great tendency to the development of local nervous disturbances in the form of subjective sensory symptoms.

The processes by which these effects are produced, whether originally of pericardial, endocardial, or myocardial origin, are twofold: diminished aspiration or suction-pump action, and reduced propulsion or force-pump action. The details of these processes have been sufficiently considered in the previous sections of this introduction, and the subject requires no further comment in this place.

PROVISIONS FOR MEETING DISTURBANCES.—Important lessons can be derived from a consideration of nature's own methods of compensating for such disturbances. The heart is in the first place endowed with a considerable reserve of energy. The extent of this margin varies doubtless within wide limits. It has been shown that the production of artificial valvular lesions gives rise to no immediate loss of blood pressure, the reserve of energy which is called up being amply sufficient to neutralise the mechanical disturbance. It has also been pointed out how impossible it is to set definite limits to this reserve.

Other more subtle arrangements are also present whereby variations in the amount of work to be done by the heart may be regulated. Although perfectly automatic in its action, the heart is—within limits—under the control of the nervous system. Among the many beautiful arrangements for the harmonious co-operation of different parts of the circulatory apparatus, which have been discussed in a previous section, there is one which may be specially recalled in this place. When the heart is labouring to overcome any obstacle to the circulation, difficult to surmount, an impression is sent to the

great circulatory nerve centres in the bulb, and is reflected therefrom to the arterial vessels, causing them to dilate and lower the pressure within the arterial circuit. In this way the outlet is increased, and the work of the heart is diminished.

When for any length of time a stress exists, it is met by the process of hypertrophy, and in this way compensation is effected. The general facts regarding this process have already been considered, and the special details receive full attention in subsequent portions of this work. It is therefore unnecessary to add anything to it in this place.

MEANS OF RELIEVING DISTURBANCES.—The most important methods of treatment in organic diseases of the circulation must be, so far as may be possible, modelled on the lines laid down by nature, although the efforts of art in this direction are but feeble attempts to follow in her footsteps.

*Rest.*—In every acute affection involving the circulation this is imperatively demanded. Hilton has emphasised the fact that, although in pericarditis complete rest is impossible, since the rhythmic movement of the heart is necessary for existence, yet through lessened action of the heart with concurrent laxness of the pericardium and diaphragm a certain amount of relief is allowed. He further points out that the exudation which takes place is a natural method of defending the serous surfaces, and that by diminishing friction it enables the serous membrane to recover itself. The influence of mechanical rest in all acute affections connected with the heart is exerted in the same direction to lessen the energy of the heart, and in this way to prevent further damage to the diseased textures, whether pericardial, endocardial, or myocardial. Mechanical rest is also a requirement of prime importance in many chronic lesions of the circulatory apparatus. In every profound disturbance of the cardiac functions whereby the hydrostatic relations are disturbed, and in such grave lesions of the blood vessels as in aneurysm of one of the great arteries, absolute rest is constantly required.

Rest produces its effects in different directions. It does not merely lessen the strain borne by damaged tissues as has just been mentioned, but it also allows the entire circulatory apparatus better opportunities of nutrition. The

amount of energy required of the heart is diminished, while the reduction of the rate of the cardiac pulsation allows not only the heart but the whole arterial system the opportunity of more repose. It is perfectly true that the blood flow through the coronary circulation takes place during the cardiac systole, and no doubt, as in the case of other muscles, the blood flow is increased by muscular activity; yet it must be allowed that the tissue changes require the alternation of activity and repose for their full performance. Rest, therefore, by prolonging the cardiac period of repose, acts beneficially in every sense.

*Sleep.*—Connected with the subject of rest is another nearly related to it, namely, sleep. This function is frequently interfered with, and diseases of the circulation produce different degrees of insomnia. Sleeplessness interferes greatly with the natural processes of repair, and the habit of disturbed or broken slumber must therefore be removed. In those instances where a condition of reflex irritability appears to be the cause of the disturbance of the sleep functions, the use of the bromides will often be found perfectly sufficient to ensure sleep. It is otherwise, however, when the lack of proper nutrition of the brain cells is the cause of insomnia. While attention to the general nutrition and to the blood pressure will, by restoring normal relations so far as possible, improve the nutrition of the brain cells, it is often necessary to use sedative measures until the healthy condition of the brain functions is restored. For this purpose a considerable number of drugs may be employed. Paraldehyde, trional, and sulphonal are all excellent in their way, but it is sometimes necessary to use others of greater power. Chloral is often recommended for this purpose, but it is a somewhat dangerous circulatory depressant, and is not often admissible. Opium and morphine are at once more certain and less risky remedies, seeing that in moderate doses they belong to the group of substances which stimulate the nervo-muscular apparatus.

*Mental Exercise.*—Also connected with the subject of rest is the consideration of the employment of the higher cerebral functions. Although any sustained mental work is necessarily out of the question, a certain amount of interesting occupation must be provided, according to circumstances.



*Air.*—The importance of an adequate supply of fresh air to any one suffering from a circulatory disorder cannot be overestimated. In the case of any one who is confined to bed, care should be taken that the chamber is thoroughly ventilated, and it is a useful matter of routine to have it furnished with two beds, one for use during the day, the other during the night. In cases where the aeration of the blood is seriously interfered with, the employment of oxygen may even be demanded.

*Light.*—The therapeutic influence of sunlight deserves also a moment's consideration, and care must be taken, as regards patients confined to bed, that their chambers have a southern exposure so as to obtain all the light available.

*Diet.*—The nature and amount of food and drink to be allowed constitute another important branch of cardiac therapeutics. Speaking generally, it is advisable that the food should be highly nutritious in quality, and diminished as far as possible in bulk. It is further to be recommended that it should be taken frequently, so that a continuous supply of nourishment may be absorbed into the circulation.

When there is too much tendency to adiposity, it may be necessary to resort to special modifications of the diet. The grounds of action here are by no means certain. According to some observers, although almost all the fat ingested is absorbed, yet but little is deposited, and the fat stored up is derived mostly from the carbohydrates and proteids. On such views Ebstein bases his treatment. They, however, are stoutly opposed by other observers.

The amount of fluid allowed to patients suffering from cardiac disease must depend upon the special conditions of each individual case. Where failure to remove fluid from the system, and a tendency to its accumulation are prominent features, the use of dry diet cannot be too strongly recommended. Its effects have been known since the days of Hope. Where, on the other hand, there is, as occurs in many cases of arterio-sclerosis with chronic renal changes and cardiac complications, a tendency to retention of waste products in the blood, the use of abundant diluents is demanded, and aerated distilled water in considerable amount will be found eminently useful.

*Digestion.*—In order to facilitate the digestion and absorption of food the state of the alimentary tract must be carefully attended to. If there be catarrh of the mucous membrane it must be got rid of by small doses of blue pill or calomel. Both of these act beneficially upon the functions of the liver through their influence upon the mucous membrane of the duodenum, and calomel has the further advantage, when given in small doses, of stimulating the functions of the kidneys. These substances may be combined with small quantities of rhubarb and some alkali, while if there be much irritability of the mucous membrane there may be also added some bismuth. Aperients may also be required, and those which are of greatest benefit in circulatory disorders are the group of saline purgatives. These are often found to act most beneficially when given in concentrated form as advocated by Hay.

*Blood.*—The condition of the blood requires attention. Hæmatinics are often of great value in the treatment of circulatory disorders. Schmiedeberg has cast some doubts upon the absorption of iron, and Bunge has attempted to prove that its beneficial effect is due to the prevention of destructive processes upon the food in the alimentary tract; Stockman, however, has shown that iron is directly absorbed. By its use in those cases presenting blood changes along with, or in consequence of, circulatory disturbances, iron, through an increase in the amount of hæmoglobin, aids the oxygenation of every tissue of the body and improves the general nutrition. Arsenic is also of much importance as a hæmatinic. Its action is still obscure, and it is impossible therefore to speak with certainty as to the mode in which it acts. It appears probable, however, that its beneficial effects are dependent upon metabolic changes and nutritive processes.

*Respiration.*—The state of the respiration must be sedulously guarded. It is of but little use to provide the patient with abundant air or even to supply oxygen, if there be any affection of the pulmonary organs by which the aeration of the blood is hindered.

*Excretion.*—The various excretory channels must be kept in activity. Elimination by means of the skin should be encouraged by the use of baths or sponging, as well as by the use of woollen

clothing. The renal secretion must be favoured according to the circumstances in existence. If there be no tendency to the retention of fluid in the system, an abundant supply of distilled water may be administered, but if there is any tendency to a water-logged condition of the system, the combination of alkalies with the cardiac tonics to be considered immediately will be found beneficial, or such drugs as caffeine may be employed.

*Removal of Fluid.*—The treatment of many cases of cardiac disease must be carried out on the lines of antihydrotic methods, as Hayem terms them, especially when there is an accumulation of serous fluid in the cellular tissue or in closed cavities—that is to say, the spaces which are regarded as belonging to the lymphatic system. This serous fluid is, as has already been shown, not lymph. It differs from it in its composition and physiological properties; particularly, it contains fewer formed elements, and has not the property of coagulating spontaneously. It is also different from exudations, due to reaction processes, which contain red and white corpuscles, and are able to deposit a considerable amount of fibrin. The one point in which the exudation resembles the transudation is that they both come from the vessels. If a large quantity of dropsical serum is injected into the peritoneum of a dog in a healthy condition, in a few hours the entire fluid will be absorbed. When, however, ascites is produced by tying the portal vein, the fluid accumulates in the peritoneum until the obstacle is removed. Hayem rightly regards all dropsies as of mechanical or of dyscrasic origin. The indications furnished are in the case of mechanical dropsies to increase the arterial pressure, and in the case of dyscrasic dropsies to improve the condition of the blood.

Sometimes when the subcutaneous tissues are very cedematous, and internal remedies have no power to bring about reabsorption of the fluid, it is necessary to have recourse to other methods of obtaining relief. This has for long been carried out by means of punctures or scarifications of the skin. Such methods are, however, attended by a considerable amount of discomfort from soaking the bedclothes with large

quantities of fluid, and the introduction by Southey of his needles and tubes is in every way a vast improvement. By means of these, the fluid is allowed to drain into receptacles instead of wetting all the clothes and bedding. Such processes require to be carried out with most rigid antiseptic precautions, in order to avoid all possibility of infection.

In many cases where the serous cavities contain large quantities of fluid, it is also necessary to obtain relief by mechanical means, through the withdrawal of fluid. From time immemorial this has been done by means of the trocar and cannula, but the introduction of the aspirator by Dieulafoy has revolutionised these more primitive means of evacuation. It is but too true that a resort to aspiration is often the prelude to the closing act of the drama. In certain cases, nevertheless, after aspiration has been carried out, recovery, not of a merely temporary, but of a more permanent character undoubtedly ensues.

Great relief is frequently obtained by bloodletting. In many cases of serious backward pressure with profound cyanosis, it is imperatively demanded, and according to circumstances it may be on a larger or a smaller scale. In many instances, the application of a few leeches will be found eminently serviceable. The amount of blood which they withdraw is inappreciable, but by the application of hot fomentations a larger amount may flow after their withdrawal. This method often gives great relief in cases even of profound hyperæmia. The process of wet-cupping is also most useful, more especially when there is any sudden and serious hyperæmia of the lungs; but beyond these processes, general bloodletting by venesection is often of the greatest utility, and sometimes rescues a patient from a state of great peril. The amount of blood to be withdrawn must be subject to the exigencies of each case.

*Exercise.*—This must be ensured in order to assist the processes of tissue change, and according to the condition of the patient may be passive or active. In the case of those unable to rise, massage must be employed along with passive movements of the limbs. The resistance exercises of Schott are also for such purposes of real importance. In the case



of persons who are able to go about, graduated active exercise is of the greatest importance, and a progressive increase in the amount employed may safely be recommended. The ascent of gentle acclivities, with frequent stoppages to recover breath, after the manner recommended by Oertel, is worthy of careful attention. These three methods of mechanical treatment—passive movements, resisted movements, and active movements—are applicable, as Lauder Brunton remarks, to different degrees of severity of heart disease.

Absolute rest for some days, followed by gentle massage and cautious passive movements, are of singular utility in the grave forms of heart disease when there is real inadequacy.

The resistance exercises introduced by the brothers Schott are fully described by Bezly Thorne, and more recently also in the works of Lauder Brunton, Broadbent, and Morison.

The resistance exercises consist in carefully regulated active movements of the different groups of muscles, gently resisted by an operator; they are never repeated twice in succession, and are always followed by a period of rest. The patient when performing these movements is carefully observed, and, if there be the slightest symptom of interference with the circulation or respiration, the movements are at once suspended. Any change in tint, whether pallor or duskiess, about the lips or cheeks, any undue dilatation of the nostrils, any contraction about the corners of the mouth, or any moisture upon the forehead, is regarded as a reason for terminating the treatment. If the patient, further, should show any appearance of weariness, such as yawning, or if he should complain of perspiration or palpitation, no further movement is executed. While the movements are carried out the patient ought to breathe regularly, and if this is not done in a perfectly natural manner, he is asked to count in whispers during the movements. While the exercises are carried out, the body and limbs should be absolutely free, so that there is no compression of the vessels.

The exercises comprise a series of nineteen movements. Each as it is performed is gently resisted by the operator. The following is a complete list:—

- (1) The arms are stretched out in front of the body at the

level of the shoulders, with the palms meeting each other. The arms are then carried outwards until they are extended laterally in line with each other, and thereafter are brought back to their first position.

- (2) The arm and hand are placed in the fully supinated position, hanging down, and the forearm is flexed upon the arm, without any movement of the latter, until the fingers touch the shoulder; thereafter the arm is extended to its original position. This movement is carried out first with one arm, and then with the other.
- (3) The arms, hanging down, are supinated, and raised outwards until the thumbs meet over the head, after which they are brought back to their original position.
- (4) The fingers of the hands, flexed at the first phalangeal joints, are pressed together in front of the lowest part of the body, and the arms are raised until the hands are above the head, after which they are brought back to their original position.
- (5) The arms, hanging in the position of "attention," are raised forwards parallel to each other until they are elevated to a vertical position, and are then brought back to that from which they started.
- (6) The body is bent forwards and then brought back to the erect position, the knees not being moved.
- (7) The body is rotated without any movement of the feet, first to one side, then to the other, and finally back to its original position.
- (8) The body is bent laterally as far as possible, first to one side, then to the other, and afterwards restored to its original erect posture.
- (9) This is a movement precisely similar to No. 1, except that it is carried out with the fists clenched.
- (10) The arms are moved in the same way as in exercise No. 2, but the fists are firmly clenched.
- (11) The arms, starting from the position of "attention," describe a circle by moving forwards and upwards

until they are raised vertically. Each palm is then turned outwards, and the arms descend backwards to their original position.

- (12) The arms, starting from the position of "attention," are moved upwards and backwards as far as can be done without bending the trunk, and are then brought back to their original position.
- (13) The patient, standing with the feet side by side and supporting himself by leaning with one hand upon any object, flexes the opposite thigh as far as it is possible, and afterwards extends it until the feet are again side by side. Thereafter leaning on the other hand, he carries out a similar movement with the other thigh.
- (14) The patient, leaning as in the last exercise, first bends the whole lower extremity of one side, kept extended, as far forwards as possible, then backwards as far as he can, and afterwards brings it beside the other. A similar movement is thereafter carried out with the other leg.
- (15) Supported by leaning both hands in front on the back of a chair, the patient flexes first one leg and then the other upon the thigh as far as he can.
- (16) Resting on one hand, the patient raises the extended opposite lower extremity outwards as far as possible, and then brings it beside its fellow. A similar movement is then carried out with the other limb.
- (17) The arms, held horizontally outwards, are rotated forwards and backwards at the shoulder joint.
- (18) The hands, held in the extended position, are first bent backwards and then forwards as far as possible, after which they are brought back to their original position.
- (19) The feet, held in their ordinary position, are first bent downwards and then upwards as far as possible, after which they are brought back to their original position.

The resistance in all cases is carried out by the operator placing the palmar surface of his hand upon the aspect of

the patient's limb towards which the movement is to be directed. In the case of the sixth, seventh, and eighth movements, the resistance is effected by the palm of the operator being placed upon the body, on that aspect towards which the movement is being carried out. The movements in the seventeenth exercise are resisted by the operator closing his thumb and forefinger round the wrist.

It is not to be understood that these movements are always carried out entirely or in the order which has been above mentioned. Some of them cannot be carried out under certain circumstances, as for instance in the case of patients who have to remain in bed, and the medical attendant in any case decides in how far it may be possible to use these movements. When these exercises are employed they must always be begun very cautiously, and with every care as regards the appearance of fatigue. While undergoing a course of exercises, the patient will require a liberal supply of food, and Schott attaches comparatively little importance to the amount and nature of the food, so long as it is nutritious. He does not lay much stress upon the amount of fluid taken during the treatment. These exercises produce considerable effects upon the circulation, as is shown by diminution of the rate, and increase in the volume, of the pulse. In addition, they lessen the area of cardiac dulness, and cause the apex beat to return towards its normal position when dilatation is present. How far these two effects are due to enlargement of the thoracic cavity cannot be determined.

Oertel's methods comprise two aims: first, diminution of the fluid contained in the body, and more especially that of the blood; and, second, correction of the circulatory disturbances and strengthening of the heart by mechanical means. The first of these consists in the reduction of the amount of fluid allowed to the patient, and the selection of a diet not containing large quantities of water. The second consists in graduated exercise, more especially in the ascent of different degrees of altitude. The changes which Oertel found to attend his system were noteworthy—the disappearance of cardiac irregularity, palpitation, and uneasiness, better filling of the arterial system, reduction of the frequency of the pulse,



increase of the energy of the heart, and absence of all respiratory distress. On percussion of the heart Oertel thought that he was able to detect a slight reduction of the cardiac dulness. With regard to the lungs the respiration became much more easy, the circumference of the thorax was increased, and the vital lung capacity augmented. The amount of renal secretion was increased, and all tendencies to œdema disappeared, whilst throughout the whole system there was a diminution of fat, shown partly by a loss of weight and a lessening of the circumference of the abdomen.

*Baths.*—The treatment of circulatory disorders by means of baths, although advocated by Hope, has not until within the last few years occupied much attention, but recently it has come greatly into favour, chiefly from the labours of Beneke, Groedel, and August and Theodor Schott. It is by no means a new discovery that the employment of medicinal waters, either internally or externally, produces a powerful influence over the circulation indirectly, by affecting the metabolic processes—many waters have for long been utilised in this way. The use of baths, however, as a means of directly affecting the circulation is of recent origin, and the method has been carefully elaborated at Nauheim.

Nauheim nestles at the foot of the Johannisberg, a small hill on the north-eastern slopes of the Taunus range in the Grand Duchy of Hesse, and is 452 feet above the level of the sea. It possesses several springs, some of which are employed internally, of which nothing need at present be said, while the others are used for the baths. The latter come from a depth of nearly 600 feet below the surface, and have a temperature varying from 82° to 95° Fahr. These waters contain from 20 to 30 parts of sodium chloride, and from 2 to 3 parts of calcium carbonate, in 1000, along with smaller proportions of potassium, lithium, magnesium, strontium, barium, iron, manganese, zinc, bromine, and arsenic. They also contain a very large amount of carbonic acid, amounting, by weight, to as much as almost 4 in 1000, and by volume 1340 in 1000. These springs rush bubbling and foaming from their outlets, one of them rising to a height of 56 feet in the air. Absolutely limpid on issuing from their sources,

they always become ruddy on standing, from the deposit of insoluble iron compounds after the escape of the carbonic acid.

As baths the waters are employed in three ways: (1) brine baths; (2) effervescing baths; and (3) effervescing current baths. Of these, the first-mentioned are employed at a strength of 15 per thousand of sodium chloride and 1 per thousand of calcium chloride, at a temperature of 94° or 95° Fahr., the carbonic acid having previously been allowed to escape. The strength is gradually increased day by day, and the temperature is modified according to circumstances. The effervescing baths contain the full amount of salts and gas, the temperature being varied to suit the individual requirements. The effervescing stream baths are the same as those just mentioned in all respects save one—the water is allowed to stream into and out of the baths while it is being used.

No one who has been to Nauheim can for a moment doubt the powerful activity of these baths under physiological conditions, or their beneficial effects in certain pathological states. The simple saline baths seem to have no influence beyond that of ordinary water at the temperature which is employed; that is to say, when the temperature is so high as to be nearly that of the body the pulse becomes somewhat more frequent and rather larger, without any perceptible change in the condition of the heart. The effervescing baths produce results very different from these. In health the effect is seen only upon the pulse, which becomes less frequent in rate, fuller in volume, and higher in pressure. At first the respirations are deeper and more frequent, but after a very short time they return to the normal. Similar effects are seen in many cases of cardiac affections, along with some reduction in the area of relative cardiac dulness. A recent visit to Nauheim afforded me an opportunity of determining these facts on myself and on others. In my own person the effects of the effervescing and the effervescing stream baths were very well marked. The pulse, which was 72 or 73 while sitting quietly in the shade in front of the bath-house, fell during the ten minutes of immersion to 61, and became at once fuller as regards its contents and larger in respect of the pulse wave.

Two hours later, after having taken a short walk and written a few letters, the rate was only 66, and the characters remained much as they were when leaving the bath. The opportunity was allowed me of examining some patients of my own, and some under the care of other physicians, and there could be no doubt of the reduction of the cardiac dulness, as well as of the increase of the cardiac energy, under the influence of the baths.

The method of employing the baths is to begin with the weak brine baths at a temperature of 95° Fahr. or thereby, the duration of the first bath not exceeding five minutes. Thereafter the bath is used with greater strength and lower temperature, while its duration is gradually increased to ten minutes. It is usual to intermit the bath every second or third day. The number of these baths varies with each individual case. As soon as the patient can stand the effervescing bath it is then administered, beginning with a temperature of from 92° to 95° Fahr. for about six minutes, and gradually employing a cooler temperature and longer immersion. If at all possible, the effervescent stream bath is employed finally. In certain cases, especially if there be any digestive or urinary troubles, the internal use of the drinking waters is enjoined, especially of the Kurbrunnen or the Karlsbrunnen. The main points in regard to these waters is, that they contain from 10 to 15 parts of sodium chloride and about 1 part of calcium chloride per mille, along with smaller quantities of other chlorides and some bicarbonates. Another spring, the Ludwigsbrunnen, is used if there be any well-marked arthritic tendencies; it has fewer chlorides and little calcium, but contains bicarbonate of sodium. The Schwalheimerbrunnen contains much more iron than the others, and is used if there is anæmia.

The Nauheim baths may be closely imitated without difficulty. In order to produce the weak bath, with which the treatment should be commenced, 1 lb. of sodium chloride and 1½ oz. of calcium chloride must be dissolved in 10 gallons of water, at a temperature of 95°. The duration of the baths should be, as at Nauheim, about five minutes. Each subsequent bath should be rendered stronger by the addition of more of

the ingredients, until the limit of 3 lb. of the sodium and  $4\frac{1}{2}$  oz. of the calcium chloride to 10 gallons of water is reached. At the same time the temperature of the bath is to be lowered, and its duration lengthened, until a temperature of  $85^{\circ}$  is reached, along with a duration of from a quarter of an hour to twenty minutes. The baths should not be given oftener than on alternate days, or on two days out of three; and it is needless to add that, as at Nauheim, the patient should lie down for some time after each bath. In about a fortnight it will usually be found that the patient is able to tolerate the effervescing baths. Sometimes, however, this period is considerably longer; it is rarely shorter.

In order to produce the effervescing baths, sodium bicarbonate and hydrochloric acid are added to the full strength of the brine bath. After having dissolved 3 lb. of sodium and  $4\frac{1}{2}$  oz. of calcium chloride in 10 gallons of water, 2 oz. of sodium bicarbonate must be thoroughly mixed with the water; 3 oz. of hydrochloric acid are then to be added just before the bath is used. This may be done by having the acid in a bottle, the stopper of which is removed at the bottom of the bath, and the acid distributed throughout the lower layer of water; or Sandow's Tablets may be used instead. The baths must be rendered more powerful day by day until 8 oz. of the alkali and 12 oz. of the acid are used for a bath of 10 gallons. The bath must be employed with the same care as that mentioned in regard to the natural baths of Nauheim, with a period of rest after each, and frequent intermissions between them.

The effects produced by such artificial baths are in all respects similar to those obtained at Nauheim. The influence of baths so prepared was carefully watched by me, when associated with Sir Thomas Grainger Stewart in the Royal Infirmary of Edinburgh; their results were investigated by us, and we were led to the conclusion, which indeed is admitted by every one, that the consequences of artificial and natural baths are identical.

It has been customary to explain the action of these baths by an increase in tissue changes produced by greater power of absorbing oxygen by the cells. Such is the explanation given



by Schott, and he pleads that it accounts for the need of rest and sleep following the administration of each bath. He holds that this increased tissue change demands great care on the part of the physician, lest an irritable and excitable state of the nervous system should be produced by excess of bathing. When injudiciously used the baths are apt to cause restlessness and sleeplessness, followed by lack of appetite and loss of strength. Schott holds that there is a reflex stimulation of the heart producing more complete and thorough contraction, as the result of which the heart becomes hypertrophied; but he is also of opinion that there may also be some direct physiological stimulation of the arterioles and capillaries by the passage of gas through the skin, so as to come in contact with the deeper tissues. Broadbent is of opinion that there is more probably a physiological dilatation of the capillaries in the skin, so that the resistance to the blood is lessened, and the left ventricle is enabled to complete its systole. In this way a more rapid transfer of blood from the venous to the arterial system would be possible. Broadbent, however, admits that the chief objection to such a view is the slowing of the pulse occurring in the bath, seeing that diminished peripheral resistance might be expected to accelerate rather than retard the pulse rate, and he throws out a suggestion that the slowing of the pulse may be attributable to reflex stimulation.

It has been said above that similar effects are produced whether the baths are natural or artificial. The effect of the exercises, moreover, must be the same wherever they are carried out, provided the operator is equally skilful. It must, nevertheless, be admitted that the results of treatment are very different when carried out in this country and at Nauheim. The reasons for this are not far to seek. The patient under treatment there is removed from the scene of his daily labours, and in most cases without doubt from numerous worries, in order to lead an existence characterised by abundance of rest and absolute quiet. The climatic conditions are usually such as to allow him to utilise the fresh bracing air of the district, and he can enjoy a large amount of sunshine, provided with sufficient shade to protect him from the direct rays of the

sun. His life at Nauheim is one of peaceful routine. He gets up between seven and eight in the morning and goes to the Wells, where, if enjoined to use the springs, he sips the water, as is usual in such bathing-places, with the accompaniment of an excellent orchestra. He then returns quietly to his hotel for breakfast, or enjoys it in some shady nook outside, and after glancing over the morning paper, has his bath. After this he lies down, and almost certainly falls into a calm sleep, from which he probably does not awake until it is time for luncheon. After luncheon he again rests until the heat of the day is past, probably sleeping part of the time, and then has the resistance movements, or some gentle exercise, until it is time for dinner, after which he is glad to seek repose. In this way, as has been frequently described to me by patients, and as my own observations have shown me, life at Nauheim is restful in the highest degree. Thus it is that there is a difference between the effects of the baths at Nauheim and any imitations of them in this country. The special indications for the use of these baths at home, or for a visit to Nauheim, will necessarily evolve from time to time in the sequel. Careful estimates of the effects of the baths are furnished by Broadbent and Morison in their recent works, and the entire system was the subject of a useful discussion introduced by Sir Thomas Grainger Stewart at the meeting of the British Medical Association in 1896. The most carefully written papers in this country, besides those mentioned, are those of Saundby and Leith.

*Special Circulatory Remedies.*—Over and above all such means of treatment, however, are the methods of acting directly upon the circulatory organs by means of drugs. The great aims of treatment as regards the effect of drugs on the heart and vessels are to modify the force and rate of the former and the fulness and pressure of the latter. By exerting influences of different kinds upon the walls of the heart and of the vessels, important effects may be produced.

There are many medicinal substances which directly affect the circulation, almost all of which belong to Schmiedeberg's great class of nerve and muscle poisons.

The first point which must be met in regard to the nerve

and muscle poisons is that their effects vary greatly, both with the amount which is given, and with the time which elapses after administration. By this is meant that many of the substances act as stimulants or tonics in small doses, while they act as sedatives or depressants in larger doses: in other words, many of them have an immediate action in one direction, and a remote effect of an opposite kind.

Another important consideration is that a distinction must be made between cardiac stimulants and circulatory stimulants. Many drugs have the power of exciting the activity of the heart, while they have no influence upon the rapidity of the circulation; many others can produce acceleration of the blood flow while possessing no effects of an exciting kind upon the heart.

It is therefore extremely difficult to classify the various nerve and muscle poisons from a purely physiological point of view, and it is certainly more convenient to arrange them in a series of pharmacological groups after the manner of Buchheim.

*The Alcohol Group.*—The alcohol group includes a large number of drugs which are important in the treatment of circulatory affections. Almost all the substances belonging to this group produce, in small or moderate doses, a dilatation of the arterioles and acceleration of the heart, and in larger doses they bring about depression of all the great vital centres, including those controlling the circulation. The researches of Zimmerberg make him doubtful if there is any direct influence upon the heart; the circulatory changes appear to him due to effects upon the nerve centres. It is, nevertheless, the general experience of clinicians that in moderate doses the alcohol series possesses considerable influence upon the heart, not only in increasing the frequency of its action—which might be due to vascular dilatation—but in causing augmented force.

Ethyl alcohol in almost any of its protean forms is of use both as a stimulant and as a food. The form which is most suitable for employment must of necessity depend upon the various attendant circumstances which accompany circulatory disturbances, more particularly upon the conditions manifested by the digestive organs. The various substances containing alcohol produce a feeling of well-being, and while in moderate doses never reaching anything which might be regarded as

approaching a tendency to anæsthesia, they nevertheless have soothing effects.

Ether acts powerfully as a stimulant, and this result is attended by sedative effects, while, given as a general anæsthetic, it may be of great use in many of the painful affections of the heart.

Chloroform, although of no benefit as a stimulant—it is, in fact, a powerful cardiac depressant—is eminently serviceable as a sedative and anæsthetic. In the form of spirit of chloroform it is most useful, since the stimulant effect of alcohol is united with the sedative influence of chloroform. It is in this way sometimes very useful in combination with other cardiac drugs given by the mouth. In angina pectoris its inhalation proves most beneficial.

Chloral is sometimes administered as a hypnotic. The large amount of chlorine in the molecule gives powerfully depressing effects to the drug. It is therefore not to be regarded as a very safe agent on account of its influence upon the vital nervous centres—this has been well shown by Harnack and Witkowski. Paraldehyde, sulphonal, and trional are worthy of much more confidence as hypnotics of this group.

The nitrites are closely associated with the alcohol group, although the characteristic action of the class is distinctly modified by special components. Spirit of nitrous ether, nitrite of amyl, and nitro-glycerin are the most important of these substances, while nitrite of sodium is a connecting link between it and another group. The special action of all these substances is dilatation of the arterioles along with a fall of the blood pressure. These drugs, which owe their introduction to Lauder Brunton, have therefore been widely employed in modern times for the purpose of reducing the blood pressure in cases manifesting too much peripheral resistance. Under the action of the nitrites the hæmoglobin of the blood, as was noticed by Jolyet and Regnard, undergoes a characteristic change from the formation of methæmoglobin, and the oxygenation of the tissues is in this way interfered with. By their antispasmodic effects on the unstriated muscle of the bronchial tubes, the nitrites are of signal use in certain forms of cardiac dyspnoea.



*The Ammonia Group.*—There is a very general belief, founded on empiricism, that the different members of the ammonia group are powerful stimulants, and although pharmacological investigation in modern times can scarcely be held to furnish any rational basis for this belief, notwithstanding the observations of Funke and Deahna, the effects observed after the administration of some of them warrant the acceptance of the empirical ideas. Ammonium carbonate and the aromatic spirit of ammonia certainly raise the blood pressure. They are therefore frequently employed, especially in combination with cardiac tonics, in many circulatory disorders. Under their influence a certain amount of vascular dilatation takes place, and the addition of such substances as the aromatic spirit of ammonia to any cardiac tonic, which is apt to give rise to contraction of the arterioles, may, in cases where such increased resistance is not desirable, be beneficially employed. It must further be remembered that in many instances, showing catarrhal conditions of the respiratory mucous membrane, excellent effects may be produced upon it by the use of the ammoniacal expectorants.

*The Camphor Group.*—The members of this class have a somewhat powerful direct stimulating effect upon the cardiac muscle, according to the observations of Heubner, and of Harnack and Witkowski, and also upon the great nerve centres, more especially in the medulla oblongata. They are therefore most useful aids in the treatment of many circulatory conditions in which there is a tendency to failure of the nervo-muscular apparatus.

*The Caffeine Group.*—Substances belonging to this group have stimulant effects, and besides exciting nerve centres, produce an increased frequency of the pulse. As will be mentioned in the sequel, they have, when taken in excess, a tendency to produce various disturbances of the nervous system, and they are therefore to be regarded as one of the many sources of functional cardiac diseases. Inasmuch as the muscle curve undergoes alterations in its character, chiefly shown by shortening of the period of muscular contraction, it is possible that these drugs act directly upon the heart. This, however, cannot be regarded as established. The substances belonging

to this group produce but little effect upon blood pressure. A very slight rise is sometimes observed and is followed by a fall. The researches of Aubert and Leven give contradictory results, and we must conclude that the effects of the group on blood pressure and pulse rate are slight. Upon the kidney the drug acts, as was shown by Brakenridge, as a diuretic, apparently by acting upon the renal epithelium. In this way it is often of much use in cardiac dropsy.

*The Morphine Group.*—The complex actions of the members of this class may often be advantageously employed for the purpose of relieving some of the effects of circulatory disease. Some of their effects are of the greatest advantage; others, however, are harmful. The beneficial effects are obtained through their actions as sedatives, anodynes, and hypnotics. The action of morphine upon the nervo-muscular apparatus in general, in small doses, is at first stimulant—in fact it raises the blood pressure; after a time, however, it acts as a depressant, producing its effects in various degrees upon the different parts of the system. Upon the sensory tracts and centres more powerful effects are produced than upon those concerned in motility, and it has therefore to be noted that there are more depressant effects upon the respiratory than upon the circulatory mechanism. In small and moderate doses the members of this group have a tendency to cause acceleration of the circulation, but in larger doses the cardiac pulsations become less frequent. It is probable that these phenomena are the result of the interferences with respiration, as suggested by Gscheidlen. The consideration of these effects shows that the drugs belonging to this class may be utilised with great advantage while proper care is exercised in their employment. In those diseases of the heart characterised by serious sensory symptoms, the various derivatives of opium are of the greatest value. In hæmorrhages due to venous stasis, the hypodermic injection of morphine may be relied upon with confidence as our most useful hæmostatic. The disturbance of the alimentary functions, and the paralytic effects upon glandular secretion, must be regarded as baneful. They are not present to the same degree after the administration of morphine as of opium. The former, therefore, is to be recommended.

*The Atropine Group.*—The substances belonging to this group have a well-marked series of effects common to all, but varying somewhat in degree and activity upon different parts of the organism. They are at first stimulants and afterwards depressants to most, if not all parts of the nervous system, and they produce these effects also upon the nervous mechanism regulating the circulation. Their effects on the circulation were very fully studied by von Bezold and Bloebaum. Upon the heart there is for a brief period a retardation of the rate of pulsation, followed by a much more marked acceleration, due to the effects produced on the vagus, while upon the vasomotor system there is probably a contraction of the peripheral blood vessels, not yet thoroughly known, so that the total effect is to produce considerable rise in pressure. Larger doses produce contrary effects. In combination with morphine, atropine is useful in certain conditions of cardiac distress.

*The Aconite Group.*—Aconite and its alkaloid have depressant effects upon the entire nervous system. Upon the circulatory mechanism there is a powerfully depressant effect, shown by frequency, irregularity, and feebleness of the cardiac contractions, along with continuous fall of blood pressure. These drugs have sometimes been employed in order to obtain sedative effects in acute diseases of the heart, more especially in endocarditis. According to my way of thinking, however, they are absolutely inadmissible in all such conditions. Aconite probably acts by dilating the blood vessels, but the work of many observers, amongst whom may be mentioned Böhm and Ringer, have not yet cleared up its mode of influencing the circulation.

*The Veratrine Group.*—At first stimulant, and afterwards depressant, in slight degree to the nervous system, veratrine, according to Kölliker, and von Bezold and Hirt, specially acts upon the muscles. The muscle curve undergoes a very characteristic alteration, the descending portion being wonderfully lengthened. Upon the heart similar effects are produced, and therefore the diastolic phase is greatly prolonged. Along with this there is a rise of blood pressure. In larger doses, the pulsations become accelerated and irregular, along

with a considerable fall of blood pressure. Used to some extent in America, veratrine has not obtained any position in this country, and it is impossible to conceive of any utility which it could possess.

*The Digitalis Group.*—There are several members of this group, first brought into prominence by Withering, which all produce similar, it might almost be said identical, effects. The effects of digitalis are mainly directed to the circulatory apparatus. An enormous literature has grown up around its actions and uses. It is impossible to do justice to all the writers on the subject, but it is not invidious to say that amongst the most important contributions are those of Vulpian, Ackermann, Dybkowski and Pelikan, Brunton, Böhm, Balfour, Schmiedeberg, Klug, and Williams. In moderate doses there is diminution of the frequency of pulsation and increased force of contraction. There is also contraction of the peripheral arterioles, and the general effect is to increase the blood pressure. It is often said that after larger doses the vascular contraction gives way to relaxation, but there is absolutely no evidence in favour of this view. Still larger doses produce irregularity and feebleness of the cardiac contractions, and the heart finally is brought to stillstand in diastole. It has been held by many authorities that the primary slowing of the heart and the contraction of the arterioles are effects due to stimulation of the vagus and vasomotor nerves, while the later effects are due to paralysis of them. Of this, however, it cannot be held that there is any direct proof, inasmuch as the same effects would be produced by the direct action of the drug upon the muscular fibres. It has been doubted whether the increase of pressure, which is admitted by all, is due to direct influence upon the arterioles, or only to stimulation of the heart. It is allowed by all that increased activity of the heart, as shown by Williams, can raise the blood pressure, but the recent observations of Lauder Brunton and Tunnicliffe may be held to prove that in addition to this there is a contraction of the smaller blood vessels.

The only member of the group besides digitalis to which attention need be directed is strophanthus, the actions of which have been carefully studied by Fraser, Polaillon and Carville,



Dybkowski and Pelikan, Ringer and Sainsbury, and Kobert. In their actions digitalis and strophanthus are very similar. They equally reduce the frequency and increase the energy of the heart, and they augment the blood pressure. The one point on which opinions differ is in regard to their effects on the arterioles. Fraser holds that they produce little or no contraction, while Ringer and Sainsbury, as well as Kobert, profess a different view. It is generally admitted that the effect on the arterioles is much less than that of digitalis.

From the therapeutic point of view, these actions of the digitalis group are almost all of advantage. By diminishing the frequency of the heart, it is allowed more rest, and along with this there is a much more thorough interchange between the blood and the muscle cells. Harnack is of opinion that too much stress is laid by physicians on this action of these drugs, but with his views it is impossible to agree. By increasing the energy of cardiac contraction, and by raising the arterial pressure, as well as by increasing diuresis, the drugs belonging to this group lessen passive hyperæmia and remove œdema, whether in the form of anasarca of the dependent parts of the body and limbs, or of effusion into the serous sacs. At the same time catarrhal conditions are removed when they depend upon passive hyperæmia.

As regards the differences in action exhibited by the various members of the group, it is unnecessary in this place to do much more than compare digitalis and strophanthus. No other member of the group is worthy of a place in the same rank as that justly accorded to these two. From long and careful observation of the actions of digitalis and strophanthus, there is no doubt left in my own mind as to their relative advantages. In cases requiring rapid effects, digitalis must be admitted to be of less value than strophanthus. Digitalis, on the other hand, is of much more importance than strophanthus in most cases presenting œdema, and more especially is this the case in mitral affections. Strophanthus is of very real importance in many instances of dilated senile heart; many examples of this condition which have been for years under my care have convinced me of its pre-

eminent usefulness in the treatment of such conditions. *Strophanthus* has much less tendency to produce nausea and vomiting than *digitalis*. It is one of the greatest drawbacks to the latter drug that so many patients find it impossible to take it on account of hyperemesis, a result extremely rare in the case of *strophanthus*. It must be admitted that idiosyncrasy alone appears to have any controlling influence upon such appearances.

An important point has lately been brought out by Deucher—that the effects of *digitalis* are much more marked when it is given subcutaneously as *digitalinum verum*. He has shown that this is due to the action of the gastric fluid on the drug when given by the mouth.

ACUTE DISEASES.—In the treatment of acute diseases some special points require to be referred to, but these can only be adverted to in general terms, as they will be fully dealt with in the chapters devoted to such special affections. The general directions with regard to rest, sleep, diet, and ventilation, which are applicable to all circulatory disturbances, are necessarily of even greater importance with regard to them. When an acute affection of the heart supervenes in the course of some general disease, such as rheumatism, the treatment which has been already in operation will require to be steadily persevered with, while in addition such further measures as the additional condition demands must be commenced. By the use of external remedies, something may be done to check the onset of pericardial and endocardial lesions. The application of ice is, in some instances, of undeniable utility. On the other hand, however, better effects are sometimes found to follow the employment of hot applications. Counter-irritation has been for long employed, but latterly Caton has suggested the repeated application of blisters, as will be more fully described in dealing with the special diseases. The use of local blood-letting by the application of leeches is also in certain of these diseases to be recommended.

With the progress of bacteriology, hopes have arisen that some system of inoculation might be found of use in acute diseased conditions of the heart. Such hopes appear to have passed from the region of expectation into the domain of

realisation, seeing that in septic endocarditis brilliant results have followed the use of antistreptococcic serum. This was used, so far as is known to me, for the first time by Sainsbury, and his results will be more particularly mentioned in the chapter on endocarditis.

**DYNAMIC DISEASES.**—In functional diseases of the circulation, the most important point is to arrive at the real cause of each affection, and it will be found, as is fully detailed in subsequent sections of this work, that very many of those disorders which are commonly classed as functional, are but expressions of some deep-seated structural modification. A large number of such dynamic or functional diseases will be found to require treatment directed towards the improvement of the cardiac tissues.

**TOXIC CONDITIONS.**—There remains, however, a considerable number of disordered conditions of the circulation owing to toxic or reflex causes, in which it is probable that little or no structural alteration has taken place. Some of these are quite transient, and disappear as soon as the influence producing them is removed. This is more especially true of those disturbances due to reflex causes. Disorders taking their origin in various poisons are not by any means so easily treated, as many of these conditions are produced by an intimate union between the poison and the tissues, sometimes, no doubt, with structural modifications, and the removal of such conditions can only be effected gradually.

## CHAPTER VI.

### CONGENITAL HEART DISEASE.

DOWN to comparatively modern times malformations were attributed to malign spiritual influences. This was indeed gravely stated by Licetus. Such ideas gradually gave place to a careful study of anatomical appearances, and the pages of Senac and Morgagni contain excellent descriptions of congenital affections of the heart. In early times antagonistic ideas were held with regard to abnormalities in development. Lémery, for example, held that such changes were due to primitive defects in the germ, while Winslow regarded them as having their origin in accidents occurring after fecundation. Concessions were made by the successors of those who held these different views, and it may be mentioned that Haller seems to have adopted an intermediate opinion.

Scientific investigations of such conditions really began with the investigations of Meckel, whose work unfortunately was marred by the doctrines of his time with regard to the development of the human embryo, the various phases of which were brought into a more or less unnatural comparison with different types of animal morphology. Bouillaud clearly enunciated the view that malformations might have a double origin; that they might be produced by diseases arising during the course of development, or might have their cause in a defective primitive constitution of the germ; he distinctly indicates that changes belonging to the former class are only to be regarded as true diseases, supervening during the period of intra-uterine life, and he would reserve the term monstrosity to original or primordial defects of the germ itself, but



he admits such an origin of malformations to be rather hypothetical. He may be held, therefore, as considering congenital malformations of the heart as due almost, if not always, to foetal diseases. Friedberg undertook a patient investigation of the development of the circulatory organs in the human embryo, and classified the malformations of the heart into groups corresponding to the three most important periods of the heart's growth. Rokitsky, in his epoch-making work on pathological anatomy, gave a complete classification of the anomalies of the heart and blood vessels. Influenced by the teaching of Dittrich, Dorsch published the result of observations upon foetal diseases, made under the guidance of his master, and in this work brought the element of foetal disease prominently forward. Chevers collected a large amount of material relating to diseases of the pulmonary artery, and Peacock, with wonderful diligence, not only reviewed most of the previous observations, but classified and criticised them, abstaining carefully from committing himself in favour of intra-uterine disease. Meyer, about the same time, analysed and grouped the many independent congenital lesions and their various combinations, attempting at the same time to attain to accurate information with regard to their causation. More recently, Heine, Kussmaul, and Lebert have filled up the gaps in our knowledge, and quite lately Thérémim has brought out the results of a long series of careful observations upon the subject.

ETIOLOGY.—The attempt to peer into the darkness which surrounds the origin of such congenital lesions is now, as it has been during the whole of modern times, a matter which has a singular fascination for the scientific inquirer. Unfortunately the facts which serve for the foundation of theories upon the subject are still defective. Now, as formerly, congenital lesions of the heart are explained by two alternative hypotheses.

There is, in the first place, what may be called the embryogenic view, which regards congenital diseases as produced by some inherent defect in development, whereby either arrest or excess of growth leads to malformation. That such a tendency exists is undoubted. It is proved by the co-

existence of other malformations in the same individual, as well as by the presence of anomalies in other members of the same family. Such a tendency is linked in the closest way with hereditary transmission. Attempts have been made to investigate the possible causes. Geoffroy Saint-Hilaire, in the early years of the present century, found that by shaking a hen's egg violently, or by coating its surface with varnish, he was able to modify the development of the chicken, and give rise to different perversions of growth. Panum and Dareste have followed out similar lines of investigation, and have been able to produce irregular forms of development by different methods of procedure. Valuable observations have also been made upon this subject by Fol, who has found that in echinoderms, if several sperm elements, instead of one, enter the germ, irregular cleavage results, and malformations follow.

Within recent years Féré has found that great modifications in development may be produced by the injection of pathogenic germs and their toxins into eggs undergoing incubation. This shows that agents, which produce disease in the individual during extra-uterine and later intra-uterine existence, give rise to the formation of malformations during the earlier phases of development. As Ballantyne so well puts it, the same causes are in action at both periods, but when influencing an embryo so far developed as to have specialised organs, the result is disease. When, on the other hand, influencing an embryo, in which such specialisation has not been carried on to the same extent, the result is a malformation. In the one case the results are pathological, in the other they are teratological. The conclusion is therefore justified that the causes of diseases and malformations are similar, if not identical, and that differences in the results are caused by different degrees of resistance in the organisms acted upon.

Antenatal life may be divided into three periods, during all of which morbid influences may affect the product of reproduction. There is, first, the period which exists previous to the union of the germ cell and sperm cell; at this time those elements, like the rest of the body, may be influenced by morbid tendencies, as is taught by everyday experience. The second period begins with impregnation, and exists until

the development of the specialised organs and the embryo, that is to say, until about the eighth or ninth week; during this time also, different morbid agents may produce modifications of growth. During both of these earlier periods the results of morbid influences are probably almost entirely teratological. The third period of antenatal existence begins with the appearance of specialised organs in the embryo, and exists until birth, and this epoch allows the development of foetal diseases instead of foetal malformations.

In tracing onwards the development of the heart as was done in a preceding section, it is possible to correlate, as Hamilton has done, different malformations with different periods of development. When the developmental processes stop at the stage when there is an auricle, a ventricle, and an aortic bulb, a malformed heart may result, consisting only of one auricle and one ventricle—one of the rarer anomalies. If an arrest takes place during the growth of the inter-ventricular septum, so that this structure becomes incomplete, a perforated septum ventriculorum results, the aperture most commonly occupying the undefended space—one of the most frequent of cardiac malformations. When a change occurs during the partition of the auricles, so that the septum is incomplete, a patent foramen ovale results, the most common of all cardiac malformations; or under other and much less common circumstances, an aperture may be left in the septum at another point, while the foramen ovale becomes closed as under ordinary circumstances. If alterations take place in the evolution of the great arterial trunks out of the aortic bulb, a number of different malformations may have their origin. A double aorta may, for example, be the result, or a communication may be left between the aorta and the pulmonary artery, or these two arterial trunks may be transposed. Still later in the process of development there may be constrictions or dilatations of the great arterial trunks, with or without the persistence of the foetal ductus arteriosus. There may, however, be minor malformations during the evolution of the complex arrangements of the branches taking their origin from the different arterial aortic arches. It need scarcely be added that at

different periods of growth duplication of any part, or even of the whole heart, may be present.

Malformations taking their origin in intra-uterine disease may be termed nosogenic, and are frequently seen. The best examples of the existence of such antenatal diseases are exhibited by the results of the infectious diseases, and by acute rheumatism. In all these cases the poisons appear to be transmitted by the mother to the child.

It has been held that the placenta affords adequate protection against poisons circulating in the maternal blood, and that micro-organisms are incapable of penetrating it. There can, however, be little difficulty in accepting it as extremely probable that the toxins produced by the activity of micro-organisms can readily transude from the maternal capillaries into those of the foetus.

The common results of such infective processes are seen in the production of endocarditis, which leads to a number of consequential changes. A stenosis of the pulmonary orifice, for instance, leads through the changes in intra-cardiac pressure, which it induces, to a patent inter-ventricular septum, to a permanent foramen ovale, to persistent ductus arteriosus, and even to a transposition of the aorta and pulmonary artery, in consequence of the septum being pushed over to the left from the higher pressure on the right side. Such are the conclusions of some of the warmest advocates of nosogenic malformations.

There are difficulties in the way of accepting the sweeping conclusions of those who would have us to believe that all congenital diseases of the heart are due to such foetal diseases. One of the greatest stumbling-blocks has been clearly shown by Osler, who points out that it is hard to suppose an endocarditis limited to the pulmonary cusps of an embryo about an inch long, whose heart could not exceed a few lines in size.

The fact is undoubted that the endocardium is peculiarly liable to intra-uterine disease, and it is of interest to note that such changes are very much more common on the right side of the heart than on the left. The pulmonary orifice is more frequently affected than the tricuspid. Lesions occurring, however, at both of these orifices produce obstruction and incom-



petence. It must be obvious that the greater liability of the right side of the heart to inflammation during foetal life arises from the relatively higher pressure and greater strain which it has then to undergo. Such lesions are very commonly associated with abnormal inter-auricular and inter-ventricular openings, and it cannot be doubted that these have their origin in disturbances in the normal pressure within the heart.

It is not without interest to note that male are apparently more liable than female children to congenital affections of the heart. It is stated, for instance, by von Dusch that of the cases he collected 64·6 per cent. were males and 35·4 per cent. females.

MORBID ANATOMY.—Some malformations are of a comparatively simple kind, consisting merely of slight variations in form and structure; but many of the changes depend upon arrest or excess in the normal processes, and lead to important structural alterations. Many of the malformations of the heart and great blood vessels render life impossible, and are therefore of comparatively little practical importance. Others do not so materially interfere with vital processes, and may be seen in those who have even attained adult age.

It is extremely difficult to classify the malformations of the heart, chiefly on account of the fact that in most instances several different structural defects are associated together. The most satisfactory, although perhaps not the most scientific method appears to lie in following out the different anomalies from the simpler to the more complex.

Displacements constitute the simplest of the modifications in the development of the heart and blood vessels. The most frequent example of this alteration is simple transposition, in which the heart bears the relation to the right side of the body which it normally ought to do to the left. The other viscera in cases of this kind are also almost always transposed, but this is not an invariable rule, some instances to the contrary having been recorded by Breschet. In such cases the heart may be perfectly well formed, as in the instance described by Samson—cases similar to which occasionally occur—or, on the other hand, various anatomical defects may be present.

The heart is sometimes situated entirely, or partly, outside of the chest. In such instances of *ectopia cordis* the organ may lie entirely external to the chest wall, it may protrude



FIG. 107.—Transposition of aorta and pulmonary artery along with patency of the ductus arteriosus and an opening through the ventricular septum.

through the diaphragm into the abdominal cavity, or it may be found in the region of the neck.

Transposition of the great arterial trunks, first observed by Baillie, sometimes occurs, the aorta taking its origin from the

right ventricle and the pulmonary artery from the left; these anomalies are usually associated with other structural modifications, as in the case figured in the accompanying illustration (Fig. 107) from a specimen described by Gordon Sanders, and placed at my disposal. It would seem no easy task to attempt an explanation of the transposition of the great arterial trunks, yet Rokitsky and Meyer have expressed some views which are not only extremely ingenious, but are also at the same time so reasonable that they may be accepted as a provisional explanation of the transposition. It has already been shown that the ventricles are separated from each other by the upward growth of the septum, which begins to make its appearance about the fourth week, and that the common arterial trunk is also separated into right and left halves by a septum taking its origin in an indentation of the walls of the vessel. The septum of the ventricles and the septum between the two halves of the arterial trunk become applied to each other, so that each ventricle is finally placed in communication with one-half of the vessel. It has to be borne in mind that the arterial end of the ventricles and the common arterial trunk undergo a rotation in the course of development, so that the portion of the trunk which finally becomes the pulmonary artery is twisted round in front of and to the left side of that which ultimately forms the aorta. Now, if through any cause this rotation is impeded at a period anterior to the coalescence of the arterial and ventricular septa, it is, as Rokitsky showed, possible for the aortic portion of the trunk to communicate with the right ventricle, while the pulmonary portion is in connection with the left ventricle. Such a cause may be present in a pericardial adhesion, as described by Meyer. In a very large proportion of instances of transposition of the great arterial trunks, there is an open septum ventriculorum, so that there is practically a free communication between both the ventricles and each of the arteries.

Malformations characterised by defect are frequent. Total absence of the heart is seen from time to time in infants who are not viable; such acardiac embryos have very commonly anomalies in other parts of the body. The pericardium is

occasionally entirely wanting, and the mediastinum, in such cases, consists only of the layers of the pleuræ with some cellular tissue. One of the earliest instances on record was described by Baillie, and examples have since been seen from time to time.

Simple malformations of individual portions of the heart may take their origin in very slight modifications of ordinary developmental processes or in intra-uterine affections. The valves, for example, which guard the orifices of the heart may differ in number from the normal standard; there may be more cusps or there may be fewer than the usual number. Such minor changes may probably owe their existence either to a faulty partition of the valvular folds, or to some endocardial disease during foetal life.

Narrowing of one or other of the orifices of the heart is found with comparative frequency. Stenosis or atresia of the auriculo-ventricular orifices may be attributed with extreme plausibility to some abnormal partition of the primitive heart during the growth of the inter-ventricular septum; it is, however, quite as probable that it has its origin in some foetal disease. Obstruction at the arterial orifices is much more frequent, particularly in regard to the pulmonary orifice. Stenosis or atresia of this channel is very common indeed, and may be seen in the pulmonary artery at its orifice, or in part of the conus pulmonalis.

To Kussmaul must be given the credit of having established the fact that a large number of the other malformations of the heart are caused by pulmonary stenosis, but, long before, Morgagni and Hunter had enunciated a similar view. This is not a theory of universal applicability. Peacock has shown that an obstruction of the pulmonary orifice from coarctation of its cusps is sometimes associated with a patent septum ventriculorum, and as the septum is completed before the valves are developed, it is difficult to understand how the causal nexus can be applied in such instances. Longstreth suggests that a reopening of the septum may take place in such cases, but to my mind it is more likely that two different causes have been at work.

The aorta and its orifices are much less commonly affected,



but the aortic orifice is occasionally found in a condition of stenosis, or the aorta itself in the neighbourhood of the ductus arteriosus may be contracted. According to Virchow a congenital narrowness of the whole aorta is found in connection with chlorosis. Abnormal width of the orifices has sometimes been recorded. The aortic orifice and aorta, for instance, have been described as enormously dilated, and this lesion has most frequently been found in association with narrowing of the pulmonary orifice and artery. Whether such changes are brought about by a developmental modification of the normal partition of the primitive aortic bulb, or produced by intra-uterine disease, is impossible to determine.

On account of absence of the internal septum, in whole or in part, the heart may consist of only two, or only three, chambers. When both inter-auricular and inter-ventricular septa are absent, the heart has been said to resemble that of the fish in plan, and when such is the case the great vessels arise by a common trunk—the primitive aortic bulb. Such bilocular hearts are incompatible with independent existence. When the inter-ventricular septum is incomplete, while the inter-auricular is more or less perfectly formed, the heart has been likened to the type seen amongst some of the reptiles. Here the blood vessels take their origin in the normal manner, and there is a possibility of existence even until the period of adult life. Instead of such grave defects there are more commonly openings through the septa. The most common of these is the persistence of the foramen ovale between the auricles.

It has been shown previously that the foramen ovale becomes, to a considerable extent, closed during the last month of foetal life, but this sometimes does not take place, and an opening, more or less guarded by a valve, may persist. According to Peacock, the opening is sometimes extremely large, and absolutely unprovided with any valvular apparatus; the aperture may be of relatively natural size, but the valvular folds may be too small to shut the opening; the valve may be of sufficient size, but it may be perforated by one or more apertures; or the valve may not be sufficiently well adjusted to close the orifice. The first three classes are not commonly

seen ; the fourth form is by far the most common malformation in the heart. It is held by some observers, amongst whom may be mentioned Longstreth, that this abnormality in the vastly preponderating number of cases results from



FIG. 108.—Patent ductus arteriosus.

obstruction at the pulmonary orifice. My own observations, however, lead me to dissent from this opinion, for a very large number of cases of patent foramen ovale are unattended by any other lesions. It is, nevertheless, often found with changes at the pulmonary or tricuspid orifice, and even with lesions of

the aortic and mitral orifices; these probably have a causal relationship by altering the course of the foetal circulation.

Partial defects of the inter-ventricular septum are most commonly found in the membranous part at the anterior end, but apertures may occur in different parts of the septum at its basal end. Two or three such openings may be present.

Some of the foetal channels between the great blood vessels, which usually become obliterated at birth, may remain permanently open. The most frequent abnormality of this kind is a patent ductus arteriosus, which is most commonly associated with other lesions to which it is probably secondary. It is shown in Figs. 108 and 109, from specimens kindly lent to me by Dr. John Thomson.



FIG. 109.—Patent ductus arteriosus.

This constitutes a malformation perfectly compatible even with adult life, and, as will be seen in the sequel, patients present themselves with the diagnostic symptoms of this defect not at all infrequently. This foetal channel, on the other hand, seems to become closed in certain cases at too early a period of development, and in consequence leads to structural alterations in order to compensate for its obliteration. It is possible that such a defect may arise from the absence of certain of the branchial arches.

Complex defects produced by combinations of the individual malformations, which have just been referred to, are more commonly met with than any of the lesions separately, with the sole exception of the patent foramen ovale. The combined changes most frequently met with are stenosis of the pulmonary orifice along with septal defect, usually of the inter-ventricular wall at its basal end. In this case it seems probable that the pulmonary stenosis is the primary factor, leading by increased pressure to interference with the normal growth of the septum. These combined defects are

often seen in association with a permanent foramen ovale, so that there is free communication between the auricles, as well as the ventricles.

It has already been seen that congenital affections of the aortic orifice are rare in themselves, and they are also uncommon in association with other defects; instances, nevertheless, are on record presenting inter-ventricular communications along with aortic atresia.

The auriculo-ventricular orifices are by no means so commonly affected, but narrowing of one or other, along with some arterial defect, is not altogether a rare occurrence. As will be mentioned in the chapter on affections of the tricuspid orifice, there is good reason for believing that obstruction at the right auriculo-ventricular opening is for the most part of congenital origin. Sometimes, associated with pulmonary atresia, tricuspid inadequacy is present, probably caused by the change at the arterial orifice.

Congenital lesions of the mitral orifice and its cusps are much rarer. A beautiful example of this defect was recently described by Carmichael. Such conditions are not infrequently associated with defective growth allowing of intercommunication between the two sides of the heart, or between the great arterial trunks; the presence of the further alteration seems to depend on the amount of interference with the blood current.

Patent ductus arteriosus in itself is rare, but it is one of the commoner malformations in association with pulmonary stenosis, and an inter-ventricular opening is often associated with these defects.

The consequences resulting from cardiac affections arising during foetal life vary widely according to the seat and extent of the changes. Many of the defects referred to render life impossible; others produce but little interference with the circulation. Speaking generally, it may be said that the mere fact of a communication between two chambers of the heart causes less disturbance to the circulation than an obstacle to the onward flow of the blood; and it is a well-known fact that, with a free communication between the two sides of the heart, there may not even be cyanosis. The usual pathological



effects, however, of the different lesions are cyanosis and various results of venous stasis as seen in the extremities and dependent parts.

Laennec believed that cyanosis was in some degree antagonistic to tuberculosis, and this opinion was taken up and more strongly urged by Rokitansky, who asserted that cyanosis provided a complete protection against tuberculisation. Since the observations of Gregory and Louis, however, it has been known that there is no truth in such assertions, and Peacock has expressly proved that there is an increased liability to tubercle in cyanotic conditions. He states that out of fifty-six patients with different forms of malformation who survived the age of eight, nine or 16·7 per cent. died of tubercular affections, while about the same period the deaths from consumption amounted to only 9·1 per cent. of the total number in the population at large. Lebert has with great diligence examined the facts bearing on this question, and has found that there is hardly a disease so commonly followed by tuberculosis as pulmonary obstruction. Duguet and Cadet de Gassicourt hold that this tendency is simply due to deficient hæmatisis.

**SYMPTOMATOLOGY.**—Just as there are frequently no general symptoms in organic disease of the heart, even in its most serious forms, so in congenital heart disease there is often an entire absence of any clinical phenomena. Cases of the kind are in consequence only detected by physical examination. An instance has been mentioned by Duroziez, in which a very large opening between the two auricles was discovered in a woman who died of erysipelas at the age of seventy-six.

The central factor upon which depends the presence or absence of general symptoms of congenital heart disease is the balance of the circulation. If there be no disturbance of the attractive and propulsive forces of the circulation, there are no outward and visible symptoms. In many cases, on the other hand, in which there is an interference with the onward flow of blood, it is possible to determine the presence of congenital heart disease at a glance.

Of the general symptoms present in congenital heart disease, that which is most prominent is cyanosis, the highest

degrees of which are only found in cases of such affections. In addition to the livid hue of the integument, which reaches a deep violet in the lips, nose, ears, fingers, and toes, more particularly in the nails, there is a characteristic prominence of the lips and nostrils. The tint of the skin always deepens with muscular exertion, and mental excitement is usually sufficient to deepen the duskiness of the skin. It need hardly be added that a paroxysm of coughing produces a great increase in the depth of the colour.

The well-known clubbing of the fingers and toes is usually present also, and the nails show a well-marked arching. Examination of the eye in almost all instances shows a sinuosity or tortuosity of the veins of the retina, which in itself is much more dusky than in health. The condition of the blood, fully described in a previous chapter, is characteristic. The specific gravity may be above 1070; the hæmoglobin may reach 160 per cent.; the number of red blood corpuscles per c.mm. may rise to above 9,000,000, and that of the white blood corpuscles to 16,000.

There is often a great tendency to hæmorrhage, which appears to be closely linked with the presence of cyanosis. The capillaries of the nose and of the gums, and even those of the respiratory mucous membrane, are apt to allow of the escape of blood.

There is also frequently a considerable tendency to transudations into the subcutaneous textures, and even into the serous sacs.

Children suffering from the effects of congenital heart affections frequently complain of cold, and shiver with the least fall of the external temperature. It often happens that the skin in such cases feels cold on applying the hand to it, and the superficial temperature, taken in the axilla, is low, while that of the mouth or rectum is usually quite normal.

Breathlessness is one of the most pronounced symptoms. There may be no dyspnœa while the child is at rest, but with any bodily or mental exertion the patient begins to pant for breath. At times there are asthmatic paroxysms quite analogous to the cardiac asthma of acquired heart disease in adults.

As a rule children born with heart lesions manifest a lethargic or apathetic condition. The mental balance remains frequently unstable for a longer period than is common in healthy children, and there is great difficulty in educating a child suffering from congenital heart disease. It is not at all uncommon to find convulsions produced as a consequence either of exertion or coughing.

The development of the entire system is retarded. The patients remain stunted, and very frequently have other malformations in addition to the cardiac disease.

An arching forward of the præcordia may often be seen. This is quite distinct from what is known as pigeon-breast, and it is probably caused by an enlargement of the heart while the walls of the chest are still very plastic.

Laennec did not have occasion to investigate the physical signs of cardiac malformations, and the first observers who attempted to systematise the means of diagnosing such conditions were Bertin and Hope.

It must be admitted that the differentiation of the various cardiac malformations is frequently attended by extreme difficulty. Some of them are unrecognisable during life, and others are so complicated as to render their absolute diagnosis impossible. When such cases are, however, discounted, there remains a certain proportion in which it is possible to determine the nature of the lesion which is present.

Entire absence of the heart may be passed over without comment, inasmuch as it is quite incompatible with the independent life of the individual. Absence of the pericardium does not seem in any instance to have given rise to clinical phenomena by means of which it could be recognised. It might, nevertheless, be reasonably inferred that some physical signs indicative of this condition should be present, analogous to those which are found in adherent pericardium from fibrinous pericarditis. Medical literature, however, contains no reference to anything of the kind.

In congenital malpositions of the heart it may occupy very various situations, and ectopia cordis may be cephalic, abdominal, or extra-thoracic. In the first and last named of these conditions physical examination reveals the position of

the organ, and in extra-thoracic cases the heart gives rise to very definite phenomena.

Simple transposition of the entire heart gives rise to physical signs by which the condition is easily recognisable. The apex beat is found on the right side of the chest, the area of dulness on percussion shows the præcordia to occupy the position on the right side which under ordinary circumstances it holds to the left, and the intensity of the heart sounds is greater on the right instead of the left side of the chest; but over and above these facts it is not to be forgotten that in cases of transposition the other organs of the body are also in almost every instance transposed. On examination, therefore, of the abdominal viscera, the liver will be found on the left side and the spleen on the right. In all cases where the malposition of the heart is present the situation of the abdominal viscera must be carefully investigated, and if they are found in their ordinary situations, every possible morbid source of acquired displacement must be rigidly excluded before it is possible to determine that the malposition is congenital.

In most of the diseases affecting its internal mechanism the pulse is empty and compressible. It is rather more frequent, as a general rule, than the normal rate. It is not uncommonly irregular. Each individual pulsation is for the most part small. The further particulars in regard to the pulse along with the other physical signs will be dealt with under the individual diseases.

*Affections of the Pulmonary Orifice.*—In obstruction at the pulmonary orifice more or less cyanosis is present, and becomes exaggerated on exertion. There may be no persistent breathlessness, but dyspnoea manifests itself with any excitement or movement. There is very commonly clubbing of the fingers and arching of the nails. The pulse is small and empty. A certain amount of prominence of the præcordia may be seen. If there are no changes in the veins or arteries of the neck, the apex beat usually occupies its ordinary situation. A thrill is often felt over the left basal portion of the heart. The cardiac dulness is enlarged to the right side, and there is a loud, usually rough, systolic murmur heard with its maximum



intensity in the second left intercostal space; the second sound in the pulmonary area is weak. The murmur is often very widely propagated, and may be heard over the entire thorax, both back and front. It is said to be occasionally conducted up to the carotid arteries; no instance of this has ever presented itself before me, and in simple uncomplicated instances of pulmonary affection it is difficult to understand by what mechanism the murmur can be so propagated.

Pulmonary incompetence is far from common amongst congenital affections. When it is present it is usually associated with obstruction, and the symptoms and physical signs are therefore due to a combination of the two lesions. In addition to cyanosis, dyspnoea, and clubbing of the fingers, there may be a diastolic thrill at the base of the heart, the right border of the heart extends farther to the right than is normal, and there is a diastolic murmur, somewhat high-pitched in its tone, which has its maximum intensity along the left edge of the sternum and about the level of the third intercostal space or fourth costal cartilage. This murmur is not widely propagated, but may be heard as far down as the xiphoid cartilage.

*Right Auriculo - ventricular Orifice.*— Affections of the tricuspid orifice are much less frequent than lesions at the pulmonary orifice, but, as will be mentioned in the appropriate chapter, it seems extremely probable that many cases of tricuspid obstruction are of congenital origin. Structural alterations at the tricuspid orifice do not produce by any means so much general disturbance of the balance of the circulation, and therefore cyanosis and dyspnoea are much less prominent features, while clubbing of the fingers is for the most part absent altogether. The pulse does not give any special features. The most characteristic physical signs of disease at the tricuspid orifice are pulsation or turgidity of the veins of the neck, a heaving impulse in the epigastric region, enlargement of the area of cardiac dulness to the right, and on auscultation, murmurs, presystolic or systolic, according as there is obstruction or incompetence. When obstruction is present a distinct presystolic thrill may be felt in the region of the xiphoid cartilage.

*Defects in the Septum of the Auricles.*—A patent foramen ovale, or other opening between the two auricles, very often produces neither general symptoms nor physical signs, and the lesion, even when on a large scale, is unexpectedly found after death even in those who have lived long lives. Several specimens exemplifying these facts are in my own possession, and my experience simply repeats that of numerous other observers. Cyanosis is not produced by defects in the auricular septum, and is only present if there be some valvular lesion, causing retardation of the blood current. Dyspnœa is also only present as the result of other lesions. It may be said, in fact, that a patent foramen ovale frequently prevents the incidence of cyanosis, and of dyspnœa, by providing to some extent a means of equalising the work of the two sides of the heart. Senac quaintly observes that when the foramen remains permanently patent it allows of prolonged diving, and even of suffocation up to a certain point. It has been held by Eichhorst that a presystolic murmur is produced by an opening between the auricles, and that this may be heard at the third and fourth left costal cartilages. If this be so, the murmur must be produced by a hypertrophied condition of one or other of the auricles, seeing that the intra-auricular pressure under ordinary circumstances is nearly equal.

Some interesting clinical features occur in cases of open auricular septum complicated by other lesions; a venous pulse in the neck, for instance, has been seen in mitral incompetence and patent foramen ovale, while cerebral embolism has not been uncommon in thrombosis of one of the veins of the leg, part of the thrombus having gained access to the left auricle by means of a patent foramen ovale.

*Defects in the Septum of the Ventricles.*—Openings between the two ventricles cause more disturbance to the general circulation, and therefore reveal themselves by more obvious clinical features than is the case with defects of the auricular septum. They may be held to produce systemic symptoms somewhat resembling those resulting from mitral incompetence, with this essential difference that the lungs escape the effects of backward pressure. It must be allowed, nevertheless, that from the stream of blood entering the right ventricle there is

augmentation of pressure and some consequent disturbance with the functions of the lungs. On excitement or exertion cyanosis and dyspnœa may make themselves manifest if they are not continuously present.

The further symptoms depend entirely upon the presence or absence of other lesions. If there be lesions at the pulmonary orifice, the symptoms produced by these lesions entirely overshadow those resulting from the patent septum. If there be obstruction or regurgitation at the tricuspid orifice, the clinical features are for the most part simply those of the tricuspid disease. In the great majority of instances of patent septum, some of these lesions are present, and the physical signs properly belonging to the septal defect are rarely met with; along with an enlargement of the cardiac dulness to the right, nevertheless, a systolic murmur has been heard in some cases at the fourth left costal cartilage close to the sternum.

*Persistence of the Arterial Duct.*—As previously mentioned, a patent ductus arteriosus is uncommon as an isolated anomaly. Instances are, on the other hand, of frequent occurrence in association with other congenital lesions.

There is usually some dyspnœa on exertion, and this is accompanied by a slight degree of cyanosis. No doubt both these symptoms are produced by the strain which is placed upon the right ventricle in consequence of the stream of blood which enters the pulmonary artery from the aorta. Interference with the voice has been seen in cases when aneurysmal dilatation of the duct has, by pressure upon the left recurrent laryngeal nerve, produced paralysis of the left vocal cord.

On inspecting the chest there is occasionally a fulness in the second left intercostal space, about an inch from the edge of the sternum, and on applying the hand over this spot a distinct thrill, accompanying or following the apex beat, may be felt. The heart on percussion is usually somewhat enlarged towards the right, but the left edge is also not infrequently farther to the left than the normal. The most characteristic physical sign is a murmur in the second left intercostal space over the septum where the thrill is felt, and this murmur is of late systolic rhythm, that is to say, that when compared with the apex beat it distinctly follows it. The murmur is usually

somewhat long in its duration, and of a high-pitched character ; it is usually louder on deep inspiration. It is further frequently accompanied by considerable accentuation of the pulmonary second sound, due to the increase of pressure in the pulmonary artery.

A change in the relative size of the pulse in the radial and femoral arteries occurs in consequence of the loss of blood in the aorta through the patent duct, and François-Franck has remarked that during deep inspiration the radial pulse becomes smaller on account of the inspiratory expansion of the lungs and aspiration of blood from the pulmonary artery.

DIAGNOSIS.—There is as a rule little difficulty in determining that a congenital heart affection is present. There are, however, instances of heart disease in the adult, as will be referred to from time to time in subsequent portions of this work, in which, although the lesions appear to have their origin in foetal conditions, absolute certainty is unattainable. Examples of such conditions are seen more frequently in connection with the pulmonary and tricuspid orifices than with any other part of the circulatory system. The differentiation of some of the congenital lesions is attended by much difficulty, more especially in young children. The limited size of the thorax, and the ease with which murmurs are conducted, render analysis of the clinical features difficult. The differential diagnosis of various congenital lesions must be conducted in accordance with the general principles applicable to heart disease which is not of antenatal origin.

PROGNOSIS.—A forecast of the future in any instance of congenital heart disease must depend for the most part upon the nature of the lesion. It must, however, be subject to considerations connected with the family tendencies. It is a well-known fact that many patients with serious congenital lesions suffer but little, while others with exactly the same condition succumb only too readily. The difference in the outlook is conditioned by the possibilities of compensatory changes, and such possibilities naturally arise from the general nutritive conditions of the individual cases.

Slight obstruction at orifices of the right side of the heart are not attended by any considerable degree of impaired health.



Openings in the septa, whether of the auricles or ventricles, are frequently found on post-mortem examination of patients who have died in advanced years; such imperfections do not necessarily interfere seriously with life, unless in the case of patent ventricular septum, where the opening is of large size.

A certain amount of obstruction of the pulmonary artery may be present without seriously interfering with vital functions. A patient under the care of Graham, for instance—described by Craigie—had been able for very hard work as a railway labourer until the age of 44, and another case was narrated by Fallot, in which the patient reached 63 years of age. On the other hand, when there is a high degree of obstruction there is not merely greater interference with functions, but there are likely to be secondary defects compensatory in their origin, and life is much shortened. Transposition of the main arteries is incompatible, as Peacock says, with the maintenance of life for any considerable period after birth.

Simple malpositions of the heart are productive of no disturbance, and absence of the pericardium causes so little interference that it is but rarely diagnosed.

The prognosis will be guided by the liability manifested by individual cases towards various symptoms. If there be a tendency, on the one hand, to dyspnœa, hæmorrhage, or convulsions, or, on the other, to pulmonary complications such as atelectasis, capillary bronchitis, broncho-pneumonia, or phthisis, the outlook is rendered by so much the more serious.

TREATMENT.—It is not an easy task to lay down definite rules for the management of congenital heart disease in general, but it may be said that this must be in every case symptomatic. In those instances which present no symptoms there can be no question of treatment, but when patients show any clinical features of heart failure in any of its forms, they should be met by appropriate means.

In the treatment of congenital disease of the heart, climate is of importance, and there can be no doubt that a dry, warm climate is best adapted to ensure the comfort and well-being of patients suffering from such diseases. The avoidance of all conditions involving mental excitement and physical exertion

is essential in cases where the circulatory equilibrium is unstable. The food must be such as will not in any way strain the digestive organs, while it must, on the other hand, be such as will afford sufficient nourishment, and render blood formation easy. A sufficient amount of regular exertion, along with fresh air and sunlight, must be enjoined, and it is also essential that the clothing, while light, should be warm. The fact has been clearly recognised since Gintrac's observations, that alcohol exercises a prejudicial influence in cases where cyanosis is a marked feature, a fact which Walshe, in his latest edition, associates with the well-known effects of alcohol under evil atmospheric surroundings. The use of the bromides has been found by me of real service in those cases of congenital heart disease where there is a tendency to erethism.

ILLUSTRATIVE CASES.—In presenting a few illustrative cases of congenital heart disease, it is unnecessary to describe instances characterised by absence of symptoms, as shown by several cases of patent foramen ovale which have been under my own observation. Those which follow are examples of unmistakable affections.

CASE 1. *Pulmonary Obstruction and Patent Ventricular Septum.*—



FIG. 110.—Clubbing of fingers in congenital heart disease.

T. K., aged eight years, was admitted to the Deaconess Hospital on October 25th, 1894, complaining of breathlessness on exertion. Both parents were alive and had always been healthy; he was one of a family of eleven, of whose members two sons had died, one from scarlet fever, and another from hydrocephalus, but the remaining six sons and two daughters were in good health.

At the time of his birth the patient was apparently a healthy infant, but, when a few months old, he became bluish in colour and had always

since been delicate. He had an attack of scarlet fever some years before admission, from which he recovered perfectly. On admission he was observed to be deeply cyanosed; the skin everywhere was of a bluish tint, the lips were almost black, and the conjunctivæ were dusky. The fingers and toes were markedly clubbed, and the nails, which were much curved, were almost black. These appearances are seen in Figs. 110 and 111. The patient was three feet nine inches in height, and weighed 2 st. 10 lb. The temperature was usually below normal, but fluctuated between 97° and 99° F. The alimentary system showed no symptoms of disturbance. The second dentition was in progress. The tongue was clean, but of a very dark purple colour. The liver exactly reached the costal margin in the right mammillary line. The hæmopoietic



FIG. 111.—Clubbing of toes in congenital heart disease.

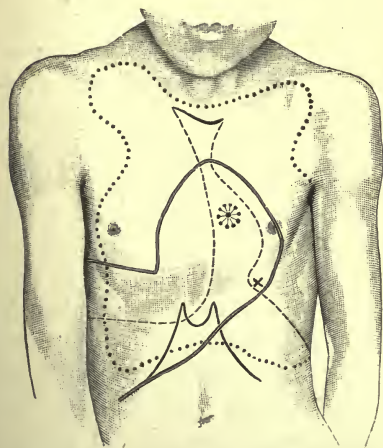


FIG. 112.—Chest tracing from Case 1. The double continuous line shows the area of dulness of the heart and liver; the line with the dashes the borders of the lungs; the dotted line the area over which the systolic murmur was heard; the cross the apex beat; and the star the maximum intensity of the murmur.

system presented some interesting facts. The spleen reached to the mid-axillary line, and was therefore of the usual size. On examination of the blood the hæmoglobin was found to be 110 per cent., the red corpuscles were 8,470,000 per cubic millimetre, while the white corpuscles numbered 12,000. The spectroscope showed the characteristic double band of oxyhæmoglobin. With regard to the circulatory system, the patient on any exertion became much more cyanosed and panted violently, but when lying quietly in bed showed much less cyanosis and almost no breathlessness. On inspection there was no visible pulsation in the neck, and in the præcordial region there was no impulse save the apex beat in the fifth left intercostal space. The pulse varied from 64 to 114.

The vessel was rather empty, the pressure low, the pulsation regular. No thrill was felt in the præcordia, but an impulse with both systole and diastole, and the apex beat was



determined to be one inch and three-quarters from mid-sternum. On percussion the borders of the cardiac dulness were found at the level of the fourth costal cartilages to be one inch and a half to the right and two inches and a half to the left of the mid-sternal line. A loud rasping murmur was heard over the entire præcordia; its maximum intensity was at the left edge of the sternum at the level of the fourth costal cartilage, and it was propagated upwards as far as the external ends of both clavicles—to the right as far as the mammilla, to the left four inches beyond the mammilla and downwards along each costal margin to the same extent, viz., two inches and a half below the level of the xiphoid cartilage. No murmur could be heard in any vessel. These physical signs are shown in Fig. 112. The præcordia were distinctly arched. There were numerous rhonchi throughout the chest, but otherwise the respiratory system presented no symptoms requiring notice. The respirations numbered 24 per minute. The urine was pale in colour and acid in reaction; its specific gravity was 1022. It contained no abnormal constituents. The integumentary system was, apart from the high degree of cyanosis, not distinguished by any special characters, and the nervous system was in all respects normal. Although the patient had never been taught, on account of his state of health, he was an intelligent and observant boy, always enjoying high spirits.

The diagnosis of the cardiac lesion was attended by some difficulty. There could be no doubt that the condition was congenital, for although the cyanosis only showed itself after the lapse of some months from the date of birth, the patient had not during the interval suffered from any acute disease capable of causing endocarditis. In this respect the history of the case was in accord with that of most congenital heart lesions. The maximum intensity of the murmur being almost in the tricuspid area might favour the view that there was regurgitation at the right auriculo-ventricular orifice; but such an explanation was negatived by the absence of any venous symptoms in the neck. On the other hand, the murmur might be produced by obstruction at the pulmonary orifice, and heard with greatest intensity over a dilated and hypertrophied right ventricle, such as undeniably was present in the case; or, again, the murmur might be the result of a communication between the two ventricles, allowing a stream of blood to flow from the left cavity to the right, and thus to produce over the right ventricle a systolic murmur. The lesions probably present, judging not only from the clinical facts of this case, but also from the experience of similar



cases, were obstruction at the pulmonary orifice and an imperfect ventricular septum. Such was the provisional diagnosis of the case.

The little patient improved considerably under symptomatic treatment, and returned to his home in the country after a few weeks' residence in the hospital. During the course of the following year, however, he died from some pulmonary affection. No autopsy could be obtained.

*CASE 2. Pulmonary Obstruction and Patent Ventricular Septum.*—M. S., aged nine, appeared as an out-patient in the medical waiting-room of the Royal Infirmary on November 25th, 1895, complaining of breathlessness. In accordance with my advice she was admitted to Ward 25. Both her parents were in good health, as were all her brothers and sisters, with the exception of one who died in infancy of scarlet fever. Ever since her infancy the patient had been of a blue tint and had suffered much from breathlessness on exertion. During the two or three weeks immediately preceding her admission to the hospital the breathlessness had become more troublesome, and the little patient had been feeling somewhat sickly.

On admission she was observed to be extremely cyanotic. The skin was of a universal bluish colour, the lips were so deeply violet as to be almost black; the ears, *alæ nasi*, fingers, and toes were also extremely dark and distinctly clubbed. The tongue was slightly furred, but there were no other symptoms of alimentary disturbance. The liver was at the costal margin in the right mammary line. The spleen reached the mid-axillary line. On examining the blood it was found to contain 95 per cent. of hæmoglobin; the coloured corpuscles numbered 6,800,000 and the colourless corpuscles 10,000 per c.mm. The spectroscope gave the double band of oxyhæmoglobin. Any excitement or any exertion produced a much greater degree

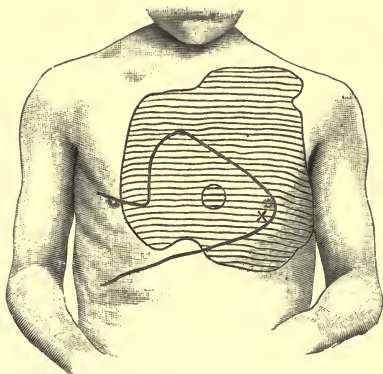


FIG. 113.—Physical signs in Case 2. The double line marks the cardiac and liver dullness, and the cross the apex beat. The area over which the systolic murmur was heard is shown by the horizontal lines, and the point of maximum intensity by the circle.

of cyanosis, accompanied by considerable dyspnoea, but when the patient lay quietly in bed, the cyanosis and dyspnoea did not cause her any distress.

The pulse was empty and compressible; its rate was about 90–100, and the pulsations were small in size. There were no abnormal phenomena connected with the neck or præcordia. The apex beat was

in the fourth intercostal space, four inches and a half from mid-sternum ; the right border of the heart was two inches, and the left border was two inches and three-quarters from mid-sternum at the level of the fourth costal cartilage. On auscultation a loud systolic murmur was heard over the entire præcordia, in fact over most of the anterior aspect of the chest, as may be seen in the accompanying illustration (Fig. 113). The maximum intensity of the murmur was at the junction of the left edge of the sternum with the fourth costal cartilage, and from this point it became gradually less distinct on passing outwards in every direction. The murmur was not conducted into the arteries of the neck.

As regards the respiratory organs, there were no symptoms with the exception of the dyspnœa. The thorax was perfectly well formed without the least tendency to bulging, and there were no physical signs of any pulmonary mischief. The integumentary and urinary systems presented no abnormality, and in regard to the nervous system the patient was a placid, contented, happy child, who slept well and was remarkably intelligent. On examining the eyes with the ophthalmoscope, Dr. Mackay described the veins as being turgid and sinuous, with a deeper tint and a broader calibre than usual.

In this case the diagnosis of the exact nature of the cardiac lesion presented the usual difficulties. As in the case mentioned previously, the position of the murmur might be held to prove that there was tricuspid incompetence, but this was negatived by the absence of any symptoms connected with the veins of the neck, and it seemed to me that in this case there was probably obstruction at the pulmonary orifice with patent septum ventriculorum, the same diagnosis, in short, as that which was arrived at in the preceding case. The little patient remained for two or three weeks in the ward, and received considerable benefit from the rest and the cardiac tonics which were administered to her.

CASE 3. *Patent Ductus Arteriosus*.—A. M., aged 26, married, was admitted on 12th September 1895, to Ward 25, then under my charge. She complained of palpitation, of pain below the left breast, of a choking sensation, and sickness. Her father and mother were both alive and in good health. One of her grandfathers had died of heart disease ; two brothers and one sister had died in infancy, and a brother had died at the age of twenty-eight of rheumatic fever. The sole remaining member of her family was a brother who enjoyed good health.

The patient had suffered from measles as a child, and had been operated on as a baby, and again at the age of seven years, for talipes equino-varus. Three years before admission she had suffered severely from palpitation. The present attack only began the day before admission, when the patient had lifted a heavy weight, and in consequence began to

suffer from the pain in the breast. She had, however, for some time previously been very alcoholic. She had no appetite, but a raging thirst, and vomited constantly. The abdominal organs presented no abnormality. The spleen reached the mid-axillary line, and was therefore of normal size. The hæmocytes were 4,500,000 and the leucocytes 9000 per c.mm. The patient complained greatly of palpitation. The pulse was a little over 100, the vessel wall was healthy and the artery was full and compressible. The pulse was absolutely regular, and the pulse wave large and bounding. On examining the neck and præcordia, nothing abnormal could be made out on inspection, but on applying the hand over the front of the chest there was a widely diffused systolic thrill, extending from the clavicles to the costal margins on both sides, and as far as the angle of the scapula on the left side behind. The maximum of this thrill was in the second left intercostal space, one inch and a half from mid-sternum. The apex beat was in the fifth intercostal space, four inches from mid-sternum, and was characterised by its extremely forcible impulse. The margins of the heart at the level of the fourth costal cartilage were two inches and a half to the right and five inches to the left of the mid-sternal line respectively. The heart was therefore considerably enlarged. On auscultation a loud rough systolic murmur was heard everywhere throughout the thorax ; in fact, it was propagated as far as the knees and elbows, but its maximum intensity was in the second left intercostal space, just at the point where the thrill could be felt most distinctly ; and on careful auscultation with the hand over the apex beat, the murmur was distinctly later than the apex beat. It was, in short, what is so commonly called a late systolic murmur. The second sound in the pulmonary area was greatly accentuated. The respiratory and urinary systems presented nothing abnormal, but the case presented features of extreme nervous excitement and insomnia.

In this case the characteristic features of patent ductus arteriosus were extremely distinct. The localisation of the thrill and murmur, the fact that the murmur was of late systolic rhythm, that it was followed by an accentuated second pulmonary sound—these points were sufficient to prove the existence of an open arterial duct. The only question in regard to which there could be any difficulty was the determination of the presence or absence of some lesion at the pulmonary orifice, and while unable altogether to negative the possibility of some slight degree of obstruction, it was clear, on account of the absence of cyanosis and other symptoms produced by pulmonary obstruction, that any such obstacle must be very slight. The lateness of the murmur, moreover, was almost sufficient to show that there was no such affection. By means of absolute rest and sedatives the patient's nervous



symptoms speedily disappeared, and she was able to leave the Infirmary in the course of two or three weeks.

CASE 4. *Patent Arterial Duct*.—E. R., aged 23, consulted me on the 23rd October 1894 on account of palpitation on exertion. The patient's family history presented no point bearing upon her condition. She had been known to suffer from congenital heart disease all her life, a patent ductus arteriosus having been diagnosed by the late Dr. Warburton Begbie when the patient was a few weeks old, and the prognosis given at that time was bad. She had, nevertheless, enjoyed tolerably good health all her life, and had passed through most of the usual infective diseases of childhood without serious trouble. The functions of the digestive system were carried out satisfactorily, and the hæmopoietic system called for no remark. The patient complained of breathlessness and of palpitation on the slightest exertion, and stated that occasionally if she had been standing a great deal, a slight degree of swelling at the ankles appeared. The pulse was full and of moderate pressure, perfectly regular, but varying considerably in rate, the limits being from 80 to 100 per minute. There were no abnormal appearances in the neck except under excitement or on exertion, when the carotid arteries pulsated violently. The apex beat was very obvious and occupied the fifth intercostal space, three inches and three-quarters from mid-sternum. A distinct thrill was felt in the second intercostal space, two inches from mid-sternum. The area of cardiac dulness was increased, and at the level of the fourth costal cartilages it reached respectively three and four inches to the right and left of the middle line. On auscultation a loud murmur could be heard over most of the præcordia, but its maximum intensity was in the second intercostal space at the point where the thrill was most distinctly felt. The murmur distinctly followed the first sound and was continued after the second, and in the pulmonary area the second sound was considerably accentuated. The functions of the respiratory and urinary systems were normal. The patient presented some symptoms of nervous excitement.

In this case also there could be no doubt of the existence of a patent arterial duct, and the fact that the murmur began at an appreciable interval after the apex beat and first sound, distinctly proved that there was no pulmonic obstruction. The administration of some tonic remedies speedily got rid of the symptoms from which the patient suffered, and she has, since the date mentioned, remained in tolerably good health.



## CHAPTER VII.

### DISEASES OF THE PERICARDIUM.

PERICARDIAL lesions were observed by Galen in the lower animals, as Senac mentions, and were apparently suspected by him in man. Clearly recognised by Lower, they received adequate description at the hands of Senac and his followers. Vieussens first called attention to adhesions between the layers of the pericardium. In more recent times, an addition to our knowledge of pathological conditions of the pericardium was made by Griesinger, who, in an unpublished lecture, referred to by Widenmann, pointed out the thickening of the pericardium and adjacent anterior mediastinum, often called mediastino-pericarditis.

Rondelet and Riverius recognised some of the symptoms produced by pericardial diseases, and these were added to by Senac, Morgagni, and others. Auenbrugger applied his method of immediate percussion to the heart and observed that, in some instances of pericardial affection, the dulness was increased; but this observation cannot be said to have been placed upon any sound basis until his translator, Corvisart, worked at the subject independently for himself. Laennec speaks of a sound like the creaking of new leather. At first he imagined this might be a sign of pericarditis, but was afterwards convinced that he was mistaken; and the honour is certainly due to Collin, who generously mentions Devilliers as having at the same time come to similar conclusions, of demonstrating that the friction sound was produced by this affection.

The connection between rheumatism and pericarditis noticed, as Wells states, by Pitcairn in 1788, was more

distinctly enunciated by Dundas, and more fully expounded by Wells and Latham.

Riolan proposed tapping the pericardium, and Senac strongly advised the operation and described how it might be done. But so far as can be ascertained, pericardicentesis was first performed by Romero.

The latest additions to our knowledge are connected with the dependence of pericarditis upon micro-organisms, and, as will be described in the sequel, Netter, Weichselbaum, Banti, and others have shown us how frequently, if not invariably, such a causal nexus obtains.

### PERICARDITIS.

There are several varieties of pericarditis. The disease may present the features of a fibrinous exudation upon the surface of the membrane, accompanied by the effusion of very little serum, or it may be essentially characterised by the large quantity of serum poured out—the fibrinous exudation preceding this being but a slight incident; the exudate may contain a large number of leucocytes, so that it possesses purulent characters; this suppurative variety, further, may be putrid; it may have a large number of red blood corpuscles, and so be fairly entitled to the term hæmorrhagic; it may be accompanied by, or depend upon, the growth of tubercle; or it may be the consequence of a carcinomatous invasion.

Some idea of the relative frequency of different varieties of pericarditis may be obtained from the observations of Breitung. Of 324 cases of pericarditis, examined in the Pathological Department of the Charité Hospital in Berlin, he found the following proportions:—

Sero-fibrinous	.	.	.	.	108
Hæmorrhagic	.	.	.	.	30
Purulent	.	.	.	.	24
Tubercular (secondary)	.	.	.	.	24
Tubercular (primary)	.	.	.	.	2
Partially adherent	.	.	.	.	111
Totally adherent	.	.	.	.	23
Ossified	.	.	.	.	2
					<hr/>
Total	.	.	.	.	<u>324</u>

The numerical relations of pericardial lesions must necessarily fluctuate within wide limits. During the last five years, according to the statistics collected by Lockhart Gillespie, there were, amongst a total of 2368 cases of heart disease admitted to the Royal Infirmary, 119 of pericarditis—68 males and 51 females, of whom 36 and 14 respectively died, *i.e.* a death-rate of 52·9 and 27·4 per cent. Of this total of 119, 68 were free from valvular complication, whose admissions and mortality have been thus tabulated:—

ADMISSIONS.						
Age.	Males.	Per cent.	Females.	Per cent.	Total.	Per cent.
1-9	1	2·5	2	7·1	3	4·4
10-19	8	20·0	11	39·2	19	27·9
20-29	12	30·0	8	28·4	20	29·0
30-39	10	25·0	4	14·3	14	20·5
40-49	4	10·0	1	3·5	5	7·2
50-69	3	7·5	1	3·5	4	5·8
+69	2	5·0	1	3·5	3	4·4
Total .	40	58·8	28	41·2	68	...
MORTALITY.						
1-9	...	...	...	...	...	...
10-19	3	37·5	3	27·2	6	31·5
20-29	5	41·6	1	12·5	6	30·0
30-39	8	80·0	2	50·0	10	71·4
40-49	3	72·8	...	...	3	60·0
50-69	3	100·0	1	100·0	4	100·0
+69	2	100·0	...	...	2	66·6
Total .	24	60·0	7	25·0	31	45·5

ETIOLOGY.—Pericarditis has a wide range of causes, which, in accordance with custom, may be grouped under the heads of predisposing and exciting agents.

*Predisposing Causes.*—The disease is certainly more common in men than women, no doubt because the primary affections to which it is consecutive are also more frequently seen in the male sex. It is essentially a disease of adolescence and early manhood. Every age, undoubtedly, may be attacked; it is, however, not a common affection either in childhood or advanced middle life; old age, nevertheless, shows a slightly increased preference for it, more especially in the presence of certain diathetic conditions. Climatic influences play a very inconsiderable part in the etiology of pericarditis; they no doubt, however, lessen the resisting power of the body by lowering the vitality. Occupation and environment act in a perfectly analogous manner, and when they are of such a character as to induce an enfeeblement of the system, they must render it more prone to the different noxious agents to be mentioned immediately.

*Exciting Causes.*—In the light thrown on diseased processes by modern methods of investigation the direct causes of pericarditis have to be regarded from a new point of view. It is therefore unnecessary to discuss the well-worn theme whether the disease is at any time idiopathic. It may be said at once that any idea of the kind is untenable. When the admitted infrequency of cases recognised as idiopathic, and the many fallacies surrounding them are taken into account, such instances as those described by Bäumler and Fagge can only be regarded as due to misconceptions. Idiopathic pericarditis has never been demonstrated. On careful investigation of all the facts belonging to any case supposed at first to be idiopathic, it is invariably found that the attack has been preceded, accompanied, or followed by some ailment, often of the most trifling description, but undeniably bearing some relation to an infective process or constitutional affection. Even those cases in which pericarditis has been attributed to direct injury can now only be considered as due to a twofold causation—external violence, and bacterial infection or chemical irritation. In all these



instances the injury, by producing a contused condition of the membrane, usually with a considerable amount of hæmorrhage, paves the way for the inroads of micro-organisms or the activities of toxic substances.

The direct causes of pericarditis naturally fall into three groups: infective processes, constitutional diseases, and direct extension.

Every acute general disease due to infective processes may be accompanied by pericarditis, and many of them are frequently its cause. Scarlet fever has, since Krukenberg first noticed the fact, been recognised as a somewhat frequent cause. Measles rarely gives rise to it, but cases have been seen by Barthez and Rilliet. Whooping cough has been observed by Racchi to be followed by the disease, and organisms obtained from the sac after death when introduced, after cultivation, into animals produced a convulsive cough. Smallpox, since the observations of Andral and Brouardel, has been well known as a cause, and erysipelas as a factor has been studied by Jaccoud. Influenza, during recent epidemics, has sometimes been complicated by pericarditis. In diphtheria it has been seen, but it is very rare. Enteric fever has often been accompanied by the affection, usually in a fibrinous form; Griesinger, however, has met with serous pericarditis, and the purulent form has been described in this disease by de Boyer. Paludism, according to Raynaud, sometimes gives rise to it. Tuberculosis often produces pericarditis, which may be primary, or which may be simply part of a general invasion of the serous sacs. Syphilis is not a common cause, but it undoubtedly gives rise to definite forms. Gonorrhœa, and gonorrhœal synovitis, are sometimes complicated by pericardial sequelæ. Septic affections, finally, are fertile sources of pericarditis, yet it is not so often found as is pleurisy in these diseases.

Of all constitutional diseases rheumatism has been known, since the time of Pitcairn, to stand pre-eminent as the chief cause of pericarditis. As a general rule, the pericardial complication appears during the second half of the first, or the early part of the second week of the disease; but, as was noticed by Graves and Stokes, the pericarditis may precede the articular symptoms. Statistics, in regard to this subject,

are not open to the serious objections which will be shown to be inseparable from those dealing with endocarditis, inasmuch as the means of diagnosing pericarditis are practically infallible, while the diagnosis of endocarditis varies with the idiosyncrasies of the observer. The best known statistics are those of Sibson, who found that of 326 cases of acute rheumatism 63 suffered from pericarditis, *i.e.* nearly one-fifth, or 20 per cent., of the total number of cases of acute rheumatism were complicated by pericarditis. This percentage, however, differs considerably from that observed by other authors; the lowest being 7·5 and 14 in the practice of Latham and von Bamberger, while much larger figures are shown by the results of such writers as Ormerod. The relative proportion of cases of pericarditis having a rheumatic origin has been often investigated. The percentages vary between 13 as given by Chambers, and 71·7 by Ormerod. Connected with this subject must be mentioned chorea, which is by no means infrequently associated with pericarditis. According to Osler it was present in 19 out of 73 autopsies which he collected, and in only 8 of these was there arthritis. Amongst constitutional diseases gout, more especially in elderly people, is frequently accompanied by pericarditis. Diabetes is at times associated with it. Some of the blood diseases usually grouped amongst affections regarded as constitutional give rise to pericarditis. Amongst these may be mentioned scorbutus and purpura. One local disease produces effects closely resembling those arising in the course of a constitutional disease like gout. This is Bright's Disease, as first observed by Taylor. According to the observations of von Bamberger, 14 per cent. of cases of Bright's Disease develop pericarditis, but Sibson only found 8 per cent., Rosenstein 7 per cent., and Frerichs 4 per cent. The affection is equally common in parenchymatous nephritis and renal cirrhosis according to Grainger Stewart, while Lecorché and Talamon hold it to be more frequent in the interstitial form of the disease. Whether in cases of gouty and nephritic pericarditis the cause lies in chemical irritation, secondary infection, or ptomaine poisoning, as suggested by Hanot in analogous cases of endocarditis, cannot yet be determined.

Pericarditis often has its origin by extension in pleurisy

and pleuropneumonia, as well as in endocarditis and myocarditis. It must not be forgotten, however, that in pneumonia the source of pericardial infection may be through organisms conveyed by the blood, and pericarditis from pneumococci has been seen without pneumonia. Septic diseases of the mediastinal glands, as well as destructive processes in the lungs, may produce pericarditis by extension, while acute affections of the bones—the sternum and ribs in front, and the vertebræ behind—have been seen to give rise to it. Septic diseases and cancerous infiltration of the œsophagus are occasionally causes. Diseases of the abdominal viscera less frequently give rise to it. Malignant pericarditis may, instead of taking its origin in direct extension, be produced by the growth of neoplasms secondary to others previously developed elsewhere.

There are certain difficulties connected with the pathogeny of pericarditis in some of these disease processes; it is not easy, for example, to understand how such a disease as enteric fever, in which the organisms very sparingly enter the blood, can produce pericardial changes. The complication must arise in such cases in one of two ways: by means of toxins formed by the organisms and carried by the blood, or by infection through another poison, for which the pericardium has been prepared by the primary disease.

Direct injury appears capable of producing the affection. No doubt in most instances it only prepares the way for the action of micro-organisms, yet it must be allowed that non-infective pericarditis may occur from violence. It has long been known that the presence of an aneurysm or a tumour may cause pericarditis, and the rupture of an aneurysm sometimes induces it, as in an instance recorded by Lee Dickinson.

Williams has narrated an interesting example of wound of the heart and pericardium, produced by a stab three-quarters of an inch to the left of the sternum, through and in the long axis of the fifth cartilage. The wound, besides involving the internal mammary vessels, was found to have produced an incision in the pericardium about one and a quarter inch in length, with a puncture of the heart about one-tenth of an



inch long, half an inch to the right of the right coronary artery and between two of its lateral branches. The wound



FIG. 114.—Fibrinous pericarditis. From a specimen kindly lent to me by Dr. Nathan Raw.

in the pericardium was closed by a catgut suture, the parietal flap was replaced, and the wound dressed. Pericarditis and pleurisy supervened. The patient nevertheless made a complete recovery, and was reported three years afterwards as being perfectly well.



MORBID ANATOMY.—As the processes involved in pericarditis pursue a somewhat similar course in all its varieties, and vary in degree much more than in kind, the most satisfactory plan in considering them is to trace out the general



FIG. 115.—Fibrinous pericarditis in a child. From a specimen kindly lent to me by Dr. Nathan Raw.

sequence of events, and point out in what particulars the different types have special features.

Pericarditis may be local or general; the former is usually seen mostly at the base of the heart, about the origin of the great vessels; both forms affect the epicardium more than the pericardium.

The earliest appearance in acute pericarditis is a dulness of the membrane, which presents a more or less ruddy tint, and often shows arborescent blood vessels, which may even have little points of hæmorrhage. Upon this a fine layer of fibrin is deposited. It sometimes presents a smooth surface, but more commonly gives rise to an appearance like that of ripple markings on the seashore, or the surface of a honeycomb, or the reticulum of the second stomach of the calf, as remarked by Corvisart; the surface frequently looks, as was said by Laennec and afterwards by Hope, as if butter had been pressed by the dairy implements known as "Dutch Hands," which were afterwards separated. This latter appearance is well seen in different degrees in the accompanying illustrations (Figs. 114 and 115). The pericardial substance is sometimes so roughened by the de-



FIG. 116.—Fibrinous pericarditis ( $\times 20$ ) showing: a, pericardium; b, epicardium; c, myocardium; d, fibrinous exudation showing organisation from both serous surfaces.

posit as to merit the term of "cor villosum hirsutumve" applied to it by the older anatomists, as mentioned by Meckel. It is of interest to note, as showing the fanciful ideas which obtained until comparatively recent times, that this appearance was, down to last century, deemed characteristic of men with bold and adventurous dispositions. There is usually a certain amount of fluid which may be almost clear, or opaque from the presence of leucocytes, or coloured from an admixture with blood.

Microscopic examination reveals layers of fibrin, felted together like the strata of a schistose rock, between the layers of which are small round cells, with leucocytes and hæmocytes. Both the epi-

cardium and pericardium show traces of infiltration by small cells. A little later organisation may be seen to proceed, as is shown in Fig. 116, from both endothelial surfaces.

In most of the septic cases, as well as in those taking their origin in acute pneumonia, masses of organisms may be seen amongst the fibrin. An invasion of the heart wall by fibrin and microbes, accompanied by the presence of leucocytes and hæmocytes, and attended by distinct changes in the structure of the muscle, is clearly to be recognised. The changes are well shown in the illustrations (Figs. 117 and 118). The cells, which are contained in the serous fluid, have their origin in proliferation of the endothelial layer.

In serous pericarditis the quantity of fluid effused varies very greatly in amount and

sometimes reaches a gallon, as in a case described by Alonzo Clark. When such is the case the pericardium is greatly distended, and on opening the fluid may form a jet, as mentioned by Radcliffe. The form assumed has been studied by means of injections by Rotch. The fluid may be clear and trans-



Fig. 117.—Acute pericarditis secondary to pneumonia ( $\times 300$ ) showing: *a*, masses of pneumococci; *b*, leucocytes, hæmocytes, and fibrin invading the heart wall; *c*, necrosed muscle fibres; *d*, healthy muscular tissue; *e*, engorged blood vessels.



parent, or slightly turbid and rather opaque. Its colour may be yellowish, greenish, or grayish, and it contains small flakes which, on examination with the microscope, are seen to be composed of fibrin. Some leucocytes and red blood corpuscles are

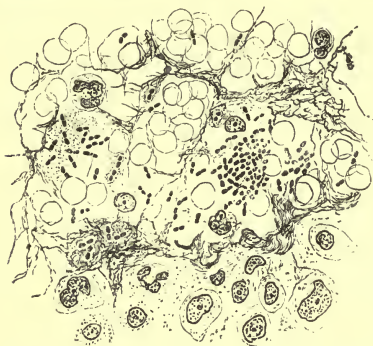


FIG. 118.—Acute pericarditis following pneumonia showing pneumococci in pairs and groups entangled amongst fibrinous meshes and associated with leucocytes and haemocytes ( $\times 1000$ ).

usually detected on microscopic examination. Sections of the epicardium and pericardium show the same changes as are observed in fibrinous pericarditis.

It is often held that the negative pressure within the pericardial sac, described in the physiological introduction, plays an important part in the production of the serous effusion, as is allowed by Hamilton. While, far from denying the possibility of such an influence, it seems

to me that, if this negative pressure played a preponderating part, a considerable quantity of fluid should be present in every case; and it is much more probable that the altered processes of the serous membranes, already described, are of greater importance. It is quite conceivable that irritants of different degrees of intensity may produce varying amounts of effusion by causing a variable effect on the capillaries.

There are some differences between the morbid appearances found in acute pericarditis as it occurs in the course of Bright's disease, and that which accompanies such a disease as rheumatism. These differences have been more especially insisted upon by Sibson. In only a small proportion of the cases of pericarditis of renal origin is there the characteristic pale rough surface formed by the fibrin, and in the great majority the exudation presents some peculiarities. There is a greater tendency to adhesion of the membranes, to the formation of pus, and the presence of blood. The pericarditis of Bright's disease may therefore be regarded as in many cases forming a transition towards the purulent, or the hæmorrhagic form.



Purulent pericarditis is essentially connected with pyæmic processes, or has its origin in some adjacent suppuration. The fluid in the pericardial sac is opaque and has a greenish yellow colour, varying in its depth according to the relative amount of fluid and cells. Sometimes the amount of fluid is very considerable, giving rise to great distension of the pericardium and displacement of the neighbouring viscera, particularly of the left lung. The epicardium in purulent pericarditis is often thick and woolly, but sometimes the plastic lymph has disappeared and there may even be ulceration into the deeper layers of the sub-epicardial tissues. The fluid, on microscopic examination, is found to contain a very large quantity of leucocytes along with some hæmocytes, and there are frequently also fibrinous flakes. Many of the leucocytes undergo fatty degeneration. A section of the epicardium shows changes closely resembling those found in fibrinous pericarditis, but there are greater structural changes in the deeper layers. In that form of purulent pericarditis which is often, on account of the decomposition of the pus, termed putrid, and which most commonly has its origin in a communication with some suppurating cavity that has been exposed to external influences, the fluid has somewhat similar characters on inspection, but is marked by its offensive odour. It teems with the organisms found in putrid suppurations, and there are frequently fatty crystals.

Hæmorrhagic pericarditis is almost always associated with a malignant invasion, as in cancer; some constitutional disorder, such as scurvy and purpura; some acute infection, such as small-pox; or a renal affection. It may be caused by an aneurysm weeping into the pericardial sac, which has been irritated by the previous pressure. Wounds of the heart of small size may act in a similar way. The amount of blood present is extremely variable. In the early stages of the affection it may be coagulated and the red blood corpuscles unaltered, but in the later stages red blood corpuscles break down and set free the colouring matter, which is then diffused through a serous effusion. In the case of aneurysms and wounds, the amount of blood may be great. The epicardium and pericardium are always deeply stained with the colouring matter of the blood,

and they frequently show extravasations. On microscopic examination there is a fibrinous deposit upon the surface of the serous membrane. This contains a large number of hæmocytes, and in some parts the endothelium and even the elastic layer beneath it are seen to be destroyed. In malignant cases, cancerous deposits may be seen with their characteristic structure.

Tubercular pericarditis is sometimes the result of localised masses in the epicardium, but at other times it is but part of an outbreak of miliary tuberculosis. In the form most commonly seen some nodules may be observed in the epicardium, sometimes threatening to invade the muscular wall. These are surrounded by fibrinous deposits and by organising tissue, in which, as shown in the illustration on p. 378, there are frequently giant cells.

The pathological consequences and terminations of pericarditis present many varieties. The most favourable termination of sero-fibrinous pericarditis is a reabsorption of the exudation and complete resolution. Instead of this, however, there are often left thickenings upon the epicardium, which give rise to the milk spots or maculæ tendineæ so often observed. There may even be larger masses of newly-formed connective tissue, which may merit the term polypoid sometimes applied to them.

Pericarditis very frequently ends in a more or less adherent pericardium. After the reabsorption of the fluid, a portion of the fibrinous exudation is in part organised by the pericardium and epicardium, from which it derives a newly-formed vascular supply; and there is in consequence a more or less complete adhesion. Partial synechia pericardii is most common at the base of the heart close to the great blood vessels. It may be composed of fine delicate bands of fibrous tissue, or of a more or less widely-spread membranous deposit. Complete synechia pericardii results in absolute obliteration of the pericardial sac, the place of which is taken by a thicker or thinner layer of connective tissue, which effects the union of the visceral and parietal pericardium.

Adherent pericardium is not, by any means, an uncommon occurrence. The statistics of Breitung, previously referred to, may be mentioned here. He found that of 324 cases of pericarditis 134, *i.e.* 41·3 per cent., resulted in adhesion.

The adhesions occasionally extend beyond the pericardium, which then becomes closely connected with the surrounding tissues. It may, in this way for instance, become attached to the anterior chest wall in front; to the œsophagus, aorta, and vertebral column behind; to the lungs and the complementary portion of the pleural sac; to the diaphragm below. Occasionally fatty degeneration takes place in the newly-formed fibrous tissue, and a deposit of inorganic salts may follow. Cases have been recorded in which there was such a degree of calcification as to leave it a matter of surprise that any movements of the heart were in any way possible. In all cases of adherent pericardium there are consequent alterations in the heart muscle. Some hypertrophy, usually with dilatation, is commonly present, and there is frequently fatty degeneration or brown atrophy of the muscular fibres. These changes will be described under the affections of the myocardium.

Sometimes the pericarditic processes extend to the pleura or to the tissues of the mediastinum, and in this way they may set up pleurisy or give rise to mediastino-pericarditis. This change results in adhesions of different kinds, and sometimes leads to suppuration. In certain cases, as originally described by Griesinger and afterwards more fully analysed by Kussmaul, the great vessels connected with the base of the heart are interfered with by means of direct or indirect attachments to the vertebral column, œsophagus, trachea, or sternum.

Occasionally in suppurative pericarditis perforation of the pericardium results, leading to the development of sinuses or abscesses, which may lead to far-reaching consequences. Many different complications occur as results of such changes.

This subject would be incomplete without referring finally to the presence of micro-organisms in connection with pericarditis. In the remarks made upon the etiology of the disease, the discoveries of many observers have been referred to; but, in order to complete the subject, the existence of micro-organisms in the fibrinous or in the serous effusion must be mentioned. Tubercular bacilli have been seen by many observers; pneumo-

cocci were first discovered by Banti; staphylococci were first seen by Wilson; streptococci by Fränkel; and micro-organisms were found by Racehi in a case of pericarditis occurring in the course of whooping cough, which, on cultivation and inoculation, gave rise to cough and pericarditis in rabbits.

**SYMPTOMATOLOGY.**—In the evolution of its clinical features pericarditis presents many different phases. Certain forms of the disease, moreover, pass through several distinct stages; some of these constitute practically the same conditions as are represented by other varieties in their fullest development, and the assemblage of symptoms is therefore at once multifarious and extensive.

*Fibrinous Pericarditis.*—Fibrinous pericarditis sometimes gives rise to no rational symptoms, but in most instances it reveals its presence by clinical features, which it is impossible to misunderstand.

Amongst the general symptoms the temperature gives extremely variable results. It is in many cases normal, or even subnormal; it is, however, in the majority of cases slightly elevated. This is more particularly the case amongst youthful patients. It sometimes, although not commonly, happens that the attack is ushered in by a rigor. The facts connected with the temperature are entirely dependent upon the origin of the affection. If pericarditis occurs as a complication of an acute disease, there may be on its occurrence a slight increase in the pyrexia already present; but it is, on the other hand, not at all uncommon to find the temperature undergoing no change, and it sometimes even falls. This latter fact is held by Paul to be relatively common in its occurrence. But his opinion is neither in accordance with the observations of most authors, nor with my own experience. If pericarditis takes its origin during the period of defervescence, or after the disappearance of the pyrexia, in any general disease, it almost invariably causes a rise of temperature.

One rather uncommon symptom in fibrinous pericarditis is dysphagia. This was noticed by Morgagni, and since his time it has been observed by several others. Sibson has placed three cases on record.



Sometimes fibrinous pericarditis is accompanied by palpitation, but this is by no means a prominent subjective symptom, although, as will be seen, the impulse becomes more forcible.

Breathlessness is occasionally present. Petit is inclined to think it a common symptom, differing, therefore, from Potain, and states that it has occasionally led him to a correct diagnosis in cases where there were no other subjective symptoms. When it is present in this affection it must owe its origin either to reflex nervous causes, propagated from the irritated serous membrane to nervous centres and reflected to the respiratory muscles, or it must be produced by extension of the lesion to the myocardium or to the lung.

Subjective sensory symptoms are sometimes present. It has already been shown that the heart in its healthy condition is not endowed with any great degree of sensibility, but the experiments of Bochefontaine and Bourceret have shown that, when there is any pericarditic lesion, the sensibility becomes greatly augmented. The nature and extent of the sensory disturbances are extremely variable. Usually there is nothing more than a feeling of uneasiness in the præcordial region, as indefinite in position as it is indistinct in character. This may be so much greater in degree as to become a sensation of oppression or of pain, even of a particularly intense kind. The position of the pain is usually præcordial, but it is sometimes experienced in the epigastric region. It may be situated in the back between the shoulders or beneath the left shoulder-blade, and it sometimes, but rarely, is found in the region of the sterno-mastoid and trapezius muscles. In a few cases there are attacks closely resembling typical paroxysms of angina pectoris, in which the præcordial pain radiates towards the left arm and is attended by all the accompaniments of that affection. Excellent examples of this have been described by Andral and by Stokes. In almost all cases where there are painful sensory symptoms there is hyperæsthesia, which indeed may be present without subjective symptoms. Nothing need be said in this place with regard to the explanation of all these appearances, since they have been fully analysed in chap. vi.

The pain at times is so severe as to produce a restrained

action of the diaphragm. Barlow more particularly brought forward this symptom as a useful diagnostic indication, and referred to the case of a boy who kept a broad belt tightly round his body in order the more effectually to restrain the movements of the diaphragm. He also mentioned another instance in which the movements of the diaphragm were much restricted, and in which no other symptom of pericarditis could be made out. Death occurred in this case, and at the autopsy the pericardium was found full of pus. In his classical lectures Hilton refers to this as a well-known symptom.

The physical examination of cases of pericarditis furnishes far more important indications than can possibly be obtained from the general symptoms; and it is the duty of the physician, in every case which is liable to be complicated by pericarditis, to examine the heart most sedulously every day.

The pulse undergoes many changes. During the onset of the affection there is often a considerable increase in its frequency, and this frequently disturbs the ordinary pulse-respiration ratio; *i.e.*, while the respiration is but slightly if at all accelerated, the pulse rate is greatly augmented. This increase in rate is not at first attended by any considerable change in the other features connected with the pulse, but after a longer or shorter period, usually very few days, the vessel becomes empty, the pressure falls, the pulsation tends to become dicrotic and irregular. These latter symptoms are probably due to extension of the lesion to the myocardium, resulting in paralytic myocarditis.

Inspection never reveals any change in the conformation of the chest due to a purely fibrinous pericardial affection, but the apex beat is sometimes observed to be more violent than in health.

When the hand is applied to the præcordia it appreciates at first a considerable increase in the force of the apex beat, and this may be, in certain cases, a useful warning, as was shown long ago by Graves. The accentuation of the cardiac pulsation, however, is of brief duration, and in a few days—sometimes in a few hours—it becomes enfeebled, probably in every case, by the spread of the process to the myocardium.

Sometimes, as Raynaud has said, the apex appears to be glued to the superficial tissues, and to crawl along under the hand.

Palpation sometimes furnishes another important sign. The friction of the epicardium against the pericardium occasionally produces a trembling vibration which is felt on palpation. This is held by Potain to be a frequent occurrence, but it is not common in the experience of most observers. This friction fremitus is at times an accompaniment of the systole; at other times it accompanies both systole and diastole. According to Potain it does not coincide exactly with the movements of systole and diastole, but is a late systolic or late diastolic phenomenon.

On percussion there is no change in the area of cardiac dulness in the early phases of the disease, but after the lapse of a few days it is often possible to determine that it is slightly enlarged on account of a degree of dilatation of the heart.

Auscultation furnishes the most important physical signs produced by pericarditis. The friction sound, heard and misunderstood by Laennec, has, since the observations of Collin, been known to be the most reliable evidence of this affection. The character of the friction murmur has been compared to sounds produced by a great many different substances. In its slighter manifestations there is a softness in its character somewhat resembling the sound produced by gently rubbing silk, parchment, or paper together. When more intense it gives the character of friction produced by rubbing two surfaces of new leather together, or of rough wood. Whatever may be its peculiar character, it invariably presents an obvious impression of being superficial. The friction sound, as first observed by Stokes, is increased on pressure, but if the stethoscope is allowed to press too heavily, it may, especially if the heart is weak, put a stop to the sound altogether. One great characteristic of the pericardial friction is that it is conducted to a very slight extent.

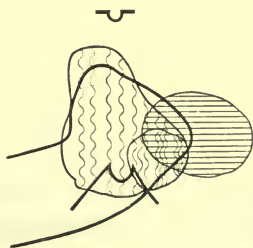


FIG. 119.—Area of audition of pericardial friction.

This is shown in Fig. 119. The slight degree of propagation manifested by the friction shows that the sound waves which are produced have but little power of setting up vibrations in the walls of the chest. The friction murmur is, however, sometimes conducted to a certain distance, and this is more particularly the case when the heart is in close contiguity to some patch of consolidation in the lung. In some cases, where a pulmonary cavity is situated close to the heart and is surrounded by consolidation acting as a means of conduction, the friction sound may be propagated into it, and may even present metallic characters, that is to say, it echoes with a metallic character within the cavity.

Pericardial friction frequently varies in loudness with the state of the respiration. The majority of authors appear to consider that it is loudest during inspiration, on account of greater displacement of the opposed pericardial surfaces during that phase of respiration. This, however, is by no means always the case, and the maximum intensity of the friction sound is not infrequently found to occur during the phase of full expiration. Potain, Traube, and Eichhorst strongly support the former as the more common occurrence, while Paul and Chabaliér consider the latter to be more frequent. This has been explained as probably due to an approximation of the pericardial surfaces through expansion of the lungs and contraction of the diaphragm. Chabaliér goes the length of stating that all those cases, in which the friction is most intense during inspiration, are connected with emphysema, and that the amount of emphysema dominates the intensity of the friction.

The position of the patient exercises considerable influence upon the intensity of the friction, which is, for the most part, heard most distinctly when the patient is sitting up, or even leaning forwards; while on lying back it sometimes disappears. The reason for this change is probably connected entirely with gravitation.

One interesting observation is that of Lewinsky, who found that, in a case where the friction was most intense during expiration, there were considerable adhesions between the pulmonic and mediastinal pleura.



The friction murmur accompanies the cardiac movements, and thus may be distinguished from that of pleurisy. Sometimes it is only heard during the systolic phase. It is, however, more commonly met with as an attendant upon both systole and diastole; and it is by no means infrequently present as a threefold sound, accompanying the auricular systole, the ventricular systole, and the diastole. This latter condition was first observed by Traube. Sometimes the friction seems to be even more broken up, the systolic or diastolic portion being separated into more or less distinct parts by intervals, as was described by Gerhardt.

The main point of interest is that the friction does not absolutely coincide with the phases of cardiac activity, than which it is somewhat later. The reason of this lack of synchronism between the pericardial friction and the cardiac sounds is exactly the same as in the case of pleural friction, which also follows the respiratory movements. A certain amount of movement of the one surface upon the other must take place before the one pericardial layer travels past the other, and in this way gives rise to the friction sound.

Paul states that in the region of the pulmonary artery the friction sound is never double; but this does not accord with my own experience, in which the sound is often not only double but even triple over the base of the heart. The sound is sometimes triple at the base and double towards the apex; while, on the other hand, these conditions may be reversed.

The duration of the pericardial friction is extremely variable. When its course is not modified by treatment, it may be present for any period of time—from a few minutes to a few hours or days—and it is to be remarked that the friction remains even after the effusion of fluid, unless this is present in great quantity.

At the commencement of an attack of acute pericarditis the heart sounds often undergo some modification in rhythm. A doubling of the first sound is the most common change, so that one form of the cantering sound is present. Potain has given an ingenious explanation of this sound. He holds that, on account of the lesion of the *épícardium*, the *myocardium* loses in part its resistance; in consequence of this the blood is

allowed to flow without obstacle into the ventricular cavity, until the moment when its repletion suddenly distends the wall. A presystolic impulse is the result, he thinks, of this sudden tension, and it is this which constitutes the first part of the canter; the normal cardiac sounds follow, constituting the two other elements in it. It is impossible to agree with this explanation, and the doubling does not in any respect require a different theory than that which has already been given.

Although the friction sound is almost pathognomonic, it is, nevertheless, sometimes, although not often, found in the absence of pericarditis. Tuberculosis and cancer give rise to a friction sound. Milk spots upon the pericardium, dryness of its surface, and ecchymoses into the subserous tissues have long been recognised as causes. Sclerosis of the coronary arteries, as in a case recorded by myself, and even hypertrophy, in the absence of all other conditions, as strongly insisted on recently by Chabaliér, may also produce a friction sound. The total number of such cases is, however, so inconsiderable that the sound may almost be considered as characteristic of pericarditis.

The course of simple fibrinous or dry pericarditis is variable. It very frequently makes its appearance in one of the general diseases previously mentioned, and entirely passes away within a few hours. It may, however, be succeeded by serous exudation and give the symptoms of pericarditis with effusion.

*Serous Pericarditis.*—When exudation of fluid takes place several characteristic symptoms make their appearance, while a change comes over some of the clinical features which have preceded the effusion.

The temperature, as a rule, undergoes but little alteration at this stage. If there has been, however, a high temperature during the earlier stage, it is, on the whole, more common to observe a fall when the fluid is poured out. In this stage, as at any other period of the affection, nervous symptoms may be present. Slight disturbances, such as headache, want of sleep, or mild, wandering delirium at night, are extremely common; while, less frequently, comatose conditions or noisy

delirium may show themselves. It is probable that such symptoms are produced by the primary condition causing the pericarditis rather than by the pericarditis itself.

Difficulty in swallowing may occur. It was studied by Festa, and was present in thirteen of Sibson's cases of rheumatic pericarditis. The special characteristics of difficulty in swallowing during the stage of effusion are that the difficulty is greatest when the effusion is at its height, and, further, that it varies with the posture of the patient. Deglutition may be extremely difficult in the recumbent position, but, when the patient sits up, the act of swallowing is rendered comparatively easy. These facts do not require a word in explanation.

The tint of the face is often extremely pale, but if there be much febrile reaction it may be characterised by a flush. Very commonly there is a considerable degree of cyanosis, and it is to be remarked that this is sometimes an extremely localised phenomenon only to be seen in the face and upper extremities. It is, however, on the other hand, very often general instead of local. Oppolzer has observed a considerable degree of cyanosis with the total absence of dyspnœa. There may be more or less œdema, which, like the cyanosis, is sometimes purely local, showing nothing but slight puffiness of the face with fulness of the neck and upper extremities. Much more commonly, however, this œdema is general and is especially found in the dependent parts. The urine is scanty, deeply tinted, and of high specific gravity. It sometimes contains albumin from venous stasis.

The breathing undergoes characteristic changes. There may be merely an increase in the frequency and depth of the respirations, that is to say, a slight persistent anhelation is present. This, however, is very frequently attended by paroxysms of dyspnœa, and it is no uncommon experience to see the patient obliged to maintain an upright position, or one in which the body leans forwards, while the respirations are laboured and frequent. These respiratory symptoms depend entirely upon, and vary according to, the amount of fluid which is present, and the rapidity with which it has appeared. In all cases where the fluid is in large quantity, the breathing is much interfered with, but it is a circumstance worthy of

attention that, when the fluid is gradually effused, dyspncea is by no means so prominent—no doubt on account of a gradual process of adaptation to new conditions. Pulmonary œdema is often present.

Hiccough is said by Peter to be present occasionally from interference with the phrenic nerve. It is possible that this symptom may be caused by pressure on the diaphragm.

Alterations in the voice have been observed. One most interesting case of this kind was recorded by Sibson, in which there was a considerable degree of aphonia. He considers that the case "tends to support the view that the left laryngeal recurrent nerve may become so affected by the contiguous inflammation as to paralyse the lungs." Similar instances have been described by Stokes and Bäumlér, but by the last mentioned observer the symptom is attributed to the pressure of the exudation upon the recurrent laryngeal nerve. Eichhorst speaks of interference also with the right recurrent nerve, and mentions that it has been considered as the result of compression by means of swollen veins.

If pain has been a prominent symptom during the onset of the affection, it usually becomes less intense and its place is taken by other sensations, which, in the absence of any previous pain, now become for the first time manifest. The sensations are of a somewhat vague character, and may consist simply in some indefinite feelings of uneasiness, weight, oppression, or sinking, in the region of the præcordia. Syncope is by no means uncommon.

The pulse, during the stage of fluid exudation, becomes of less volume and lower pressure. It is, as a rule, fully dicrotic; irregularity, as well as inequality, may often be determined; the bigeminal, or alternate, or even paradoxical pulse may be found. Traube observed the left radial and carotid pulses smaller than the right in one case, and the same was seen, along with dilatation of the left pupil, by Smith. Such symptoms denote pressure on the arterial trunks and sympathetic system. The veins of the neck undergo, in many instances, a considerable degree of distension; or undulations may be seen in them. According to Friedreich these venous movements are definite regurgitant



waves, but Riegel has shown with justice that they are simply from the auricular contractions. A real venous pulse in pericarditis speaks for an accompanying tricuspid incompetence. An inspiratory swelling of the veins of the neck is at times seen. These appearances are more especially associated with cyanotic symptoms.

All these symptoms are the direct result of the fluid in the pericardium, as Lower clearly showed two centuries ago. By its presence it compresses the lungs, and more particularly the left lung; while it also lessens the movements of respiration. It, at the same time, interferes more especially with the diastolic movements of the heart, and by both processes it interferes with the oxygenation of the blood. The cyanosis and œdema are the direct results of backward pressure. Experimental proof of this has been furnished by the observations of Cohnheim, which were previously described, and which have been repeated by Starling.

The fact must not be overlooked that some of the symptoms may not be altogether dependent upon mechanical interference with the heart from the presence of the fluid, but may take their origin in a paralytic myocarditis, produced either by extension, or by identical etiological factors, affecting the myocardium and pericardium simultaneously. The least resisting portions of the heart—the auricles—are mostly interfered with by the presence of fluid; hence the venous stasis, not only in the systemic vessels, but also, and perhaps more particularly, in those of the lungs. In this way œdema and cyanosis are accounted for.

On inspection of the præcordia a fulness or even a distinct swelling may be seen. The presence or absence of such a bulging does not altogether depend upon the amount of fluid which is present; several other factors are concerned in its appearance. It is, for instance, by no means such a prominent feature in those who are well nourished, as in those who have a spare habit of body. It therefore happens that certain cases present this symptom, although a comparatively small amount of fluid is present; while, on the other hand, there may be no arching forward in other instances where a large amount of fluid has been effused. When such a bulging

is present, it is usually found along the left edge of the sternum, extending from the third to the fifth rib; but it is sometimes more extensive and may occupy the space existing between the second and the sixth ribs.

The impulse of the heart becomes less distinct, and it may be impossible to see it. The apex beat is more mobile, in some cases, from the enlargement of the sac. Skoda, who has been followed by most authors, held that the fluid, being of less specific gravity than the heart, in a recumbent posture occupies the anterior part of the sac. That this is erroneous has recently been shown by Rehn and Schaposchinkoff.

On palpation the cardiac impulse, which has been described as exaggerated in the stage of fibrinous exudation, becomes less and less distinct after the fluid has made its appearance, until it usually becomes, as has just been said, so feeble as to be inappreciable to touch. The vocal fremitus over the sternal region loses some of its intensity. Fluctuation has sometimes been observed.

The most characteristic symptoms in pericarditis with effusion are elicited by percussion. Skodaic resonance may be heard over the adjacent lung tissue from its relaxed condition. The area of cardiac dulness steadily increases during the process of fluid exudation until it attains its maximum, and, when this has occurred, it begins, after a longer or shorter interval, to diminish. The increase in the cardiac dulness takes place in every direction, and it affects both the deep, or relative, and the superficial, or absolute, dulness. Rotch has stated that in an early stage dulness may be elicited in the fifth right intercostal space. This cannot be depended upon. The extent of the relative dulness necessarily depends altogether upon the amount of the fluid, and the form which it assumes is, in cases where it is at all extensive, extremely characteristic, as was shown by Sibson. Its outline has been compared, sometimes aptly but much more often inaptly, to various objects. It, in almost every instance, has an outline closely resembling that of a pear hanging downwards by its stalk. The narrow upper portion of the cardiac dulness forms, as Sibson showed, a peak; and, at the upper and left boundary of the dull area, there is an indentation. This characteristic outline may be modified by

pericardial adhesions, or by some adjacent affections; it is, nevertheless, an extremely useful point in diagnosis, in spite of the adverse opinion of Shattuck. In certain cases there is a patch of dulness, as shown by Ewart, over the lower thoracic region behind.

The features, ascertained on auscultation, are to a considerable extent conditioned by, but are not altogether dependent upon, the amount of fluid. The vocal resonance over the lower part of the sternum, like the fremitus, wanes in intensity. As was noted by Gendrin, the auscultation of the voice over the præcordia, while the patient is in a sitting posture, sometimes reveals the presence of ægophony. Sometimes there is bronchial breathing in the infra-mammary and infra-scapular regions, to which Ewart has called attention. The heart sounds always become feebler; partly, no doubt, from the presence of the fluid, which not only lessens the afflux of blood and so interferes with the suction-pump functions of the heart, but also interferes with its driving powers. In many cases, however, the weakness of the heart sounds must be, in part at least, produced by myocarditis extending inwards from the affected surface.

The friction sound sometimes disappears on the advent of the effusion, but in many cases it remains throughout the whole course of the disease. It has been found by Čejka to persist even when the sac contained 1000 c.cm. of fluid. This may sometimes be due, no doubt, to the small amount of the effusion; but it is also probable that, in those cases where a considerable amount of effusion has occurred, some fibrinous attachments may have been caused. In every case, however, in which the friction murmur persists, it becomes considerably weakened after the effusion has set in. In cases previously presenting no endocardial murmurs, the development of systolic mitral and tricuspid murmurs is common from weakening of the myocardium.

The usual result of serous pericarditis is more or less complete resolution. The fluid is reabsorbed, and even the fibrinous exudation may undergo the same process, leaving the membrane almost intact. Not uncommonly, however, a certain amount of organisation takes place, and some adhesions are formed between the pericardium and epicardium. In such cases symptoms of

obliteration of the sac to a greater or lesser degree are gradually developed. There may, on the other hand, be a change in the contents of the sac, and purulent or hæmorrhagic pericarditis may then possibly be detected. But, over and above all these contingencies, the morbid process may become arrested from lack of tissue reaction; in such an eventuality chronic pericarditis is the result.

*Purulent Pericarditis.*—The clinical features in purulent pericarditis present differences according as the formation of pus has been present from the first or has occurred in consequence of an alteration in the type of the morbid process.

Sometimes when pericarditis has been from the first characterised by purulent exudation, it may be so masked by the preceding or accompanying conditions as to have scarcely any recognisable clinical features. When it occurs, for example, in the course of a septicæmic affection, there may be no alteration in the general conditions, and the only possibility of recognising the pericardial complication lies in the presence of physical signs. Even these may fail in certain instances, as in Case 9, which is described on p. 356. In cases of this kind, nevertheless, the accelerated, feeble, and often irregular pulse may call attention to the probability of a pericardial complication.

In those cases of pericarditis, however, which form the principal, and sometimes the earliest manifestation of invasion by pyogenic organisms, and which may therefore be termed primary purulent pericarditis, the symptoms are more characteristic. In such instances well-marked rigors are common. The temperature is as a rule high, and subject to the characteristic fluctuations seen in septic diseases. The pulse is frequent but variable, and the respiration is accelerated. Profuse perspirations are also of common occurrence. It must nevertheless be admitted that purulent pericarditis may be found with a temperature almost normal, and occasionally even subnormal, along with but little alteration in pulse and respiration.

The physical signs, further, in purulent pericarditis, present many varieties. Considerable acceleration of the pulse, as has just been mentioned, is common, and the vessel is empty,



while the pressure is low. High degrees of dicrotism are also frequently present, while irregularity is common. There may be no characteristic appearances on inspection of the præcordia, or there may be a degree of prominence accompanied by disappearance of the apex beat, which may be imperceptible on palpation. On percussion the area of cardiac dulness is increased; but as will be seen in the case described below, when mediastinal abscesses are present it may be impossible to determine this point. On auscultation a friction sound may be heard; but here again the element of difficulty is sometimes present, inasmuch as, from beginning to end, pericardial friction may never be developed.

When an exudation, previously of a simple serous type, becomes purulent, it is a matter of greater ease to arrive at correct conclusions. In a case of this nature there is almost invariably shivering, followed by characteristic fluctuations of temperature, pulse, and respiration, accompanied by excessive perspirations and the development of a hectic condition with rapid wasting and great prostration. The physical signs do not necessarily undergo any change, and in many cases manifest no modification on the transformation of the exudation. The pulse, however, has an undeniable tendency to become irregular and dicrotic, while the extent of cardiac dulness may be increased. In such cases as these it is the duty of the physician by aspiration to determine the character of the exudation.

*Hæmorrhagic Pericarditis.*—The exudation in pericarditis contains blood in a considerable proportion of cases, more particularly when it occurs in the course of an already existent cardiac or renal disease. In old age, as well as in chronic cases of alcoholism, a sanguineous exudation is apt to occur, and in the course of tubercle and cancer of the pericardium it is common. Beyond these causes blood may occur in the pericarditis which complicates some cases of the hæmorrhagic eruptive fevers, and in scorbutus and purpura it almost constitutes a special affection.

Sometimes the phenomena present in such cases present but little that is characteristic, but if there be any considerable degree of hæmorrhage, it reveals itself by phenomena

resembling those of severe hæmorrhage in general; that is to say, a tendency to vertigo, oppression in the chest, cold perspirations, irregularity and emptiness of the pulse, chilliness of the extremities, and syncope, often leading to sudden death.

The occurrence of such symptoms in the course of any disease likely to lead to hæmorrhage might justly lead to the suspicion that the pericardium was implicated.

*Chronic Pericarditis.*—A word or two must be added with regard to chronic forms of the disease. As a rule, chronic pericarditis is simply the remains of an acute pericarditis, in which resolution has been impeded at one of its stages, but there are cases in which from the first the disease is chronic. This is more particularly the case in old people, as well as in those who are alcoholic, or who suffer from kidney disease.

In the case of an acute pericarditis arrested during the progress of resolution, the clinical features presented are simply those of the stage at which the disease has arrived when the arrest occurs. When, however, the disease is from the first chronic, it very commonly pursues an entirely latent course. There may be weakness of the circulation and interference with the breathing. Sometimes there is a sense of oppression in the præcordia produced by free effusion; pain is very rarely complained of.

On examination of the chest in such cases the physical signs are:—an enlarged area of cardiac dulness; weakness of the heart sounds; sometimes a friction murmur. In the absence of the friction sound the condition may closely resemble hydropericardium, more especially when the course of the disease has been insidious.

**DIAGNOSIS.**—The recognition of pericarditis is not in the great majority of cases a matter of difficulty, although it must be allowed that some of its forms may either be latent, or so masked by other conditions, as to render detection difficult. The general symptoms are seldom so characteristic as to be of great value in the diagnosis of pericarditis, and even the physical signs may be misleading, as, for example, in case No. 9, on p. 356.

It might at first sight seem improbable that the friction sound could be mistaken for any other phenomenon revealed

by auscultation ; yet, in addition to the possibility of its being generated by pericardial roughness or dryness previously referred to, there are dangers of misinterpretation. The exocardial friction sound may, in the first place, be mistaken for an endocardial murmur. It is, as has been seen, usually louder on inspiration than on expiration, while the endocardial murmur is as a rule of less intensity during the inspiratory phase of respiration. As shown by Traube, however, endocardial murmurs are occasionally rendered louder by inspiration, and the general principle is, therefore, subject to considerable variations, inasmuch as the friction sound may be louder during expiration.

The friction sound is of a more superficial character, and a more grating quality. It is rarely propagated beyond the præcordia, and is usually increased in intensity by gentle pressure with the stethoscope, while an endocardial murmur undergoes no change with such increase of pressure. It is sometimes remarked that both exocardial and endocardial sounds are weakened if the movements of the heart are interfered with by too great external pressure. The position of the patient is occasionally, but by no means commonly, of assistance, at any rate in distinguishing between systolic endocardial murmurs and any external friction sound. Systolic murmurs are almost invariably louder while the patient is in the recumbent posture, while the exocardial friction sound is usually loudest while the patient sits up or even leans forwards. According to Gerhardt and Bernheim, if an adventitious sound is only heard when the patient sits up it is always pericardial. This is far from being the case—many presystolic mitral murmurs are only rendered audible in an upright posture.

On carefully attending to the rhythm of any exocardial murmur, a distinction can be made out between it and endocardial murmurs, because, as has been seen previously, the friction sound follows with a slight interval, or at least does not accurately coincide with, the cardiac sounds. After it has been determined that a friction sound is present, there is still, in the second place, a possibility of misinterpretation. The pericardial may be mistaken for pleural, or even peritoneal, friction ; that is to say, an error may be made in the localisa-

tion of the physical sign. Such an error is most likely to occur in connection with the pleural pericardium than any other part. It is not uncommon to find a friction sound in pleurisy over that part of the left lung which projects forward at the inferior border of the absolute cardiac dulness. The part of the pleura, which encloses this small projecting portion of the lung, is necessarily in intimate connection with the heart. Pleurisy in this region presents difficulties in consequence of the fact that the friction sound accompanies the movements of the heart as well as the movements of respiration. On deep inspiration a loud friction sound may be heard, and if the patient be made to hold the breath in this phase of respiration, every trace of the friction sound will disappear; but if, on the other hand, the breath is held at the end of expiration, a distinct pericardial friction sound will be heard, which, however, dies out after two or three pulsations have taken place.

A friction sound has been described in tubercular peritonitis, produced, in consequence of the movements of the heart, by the rubbing of the rough surfaces of the diaphragmatic peritoneum and that investing the liver. Emminghaus has carefully studied the occurrence of this physical sign, which is certainly very rare.

Difficulties in diagnosis may also be present after fluid effusion has occurred. The increased area of cardiac dulness may be mistaken for a heart enlarged by dilatation, or for the presence of hydropericardium. There is also the possibility of error from its being taken for mediastinal tumour or abscess, or for thoracic aneurysm. Cases, like those of Smith, with differences in the radial pulses and in the pupils may be extremely like instances of aneurysm. Consolidation of the lung, or localised and encysted pleurisy may even be a cause of error. The characteristic form of the area of dulness should render the diagnosis easy, yet it may be modified by pericardial adhesions, or by the presence of some accompanying affection. In pyæmic cases a dull area of large size may be produced by the presence of an abscess along with pericarditis.

In most instances the conditions under which pericarditis arises will prevent the possibility of its being mistaken for any of these conditions, while the mode of onset and physical signs of tumours, aneurysm, pulmonary consolidation, or



encysted pleurisy, ought to obviate any possibility of misconception. Some forms of pericarditis may readily enough be mistaken for hydropericardium; in the latter condition, nevertheless, the state of the circulation, or the condition of the kidneys, ought to furnish reliable indications. The difficulty cannot be so readily solved as in the analogous difficulty between pleurisy and hydrothorax, since it cannot be considered justifiable to explore the pericardium with the aspirating needle unless it be urgently called for by the condition of the patient.

PROGNOSIS.—The prognosis in pericarditis depends largely upon the personal characteristics of the patient. It is, for example, much more serious during early childhood and again in advanced years. It is, further, more fatal in women than in men; but, over and above the factors of age and sex, the individual peculiarities of the patient exercise considerable influence.

The existence of any affection, such as renal disease, by means of which metabolic functions are retarded, renders the outlook less favourable, while alcoholism and exhaustion, whether from excess or fatigue, are to be regarded as reasons for caution in formulating the prognosis. The condition of the valves and of the cardiac muscle have to be taken into account. If, from causes connected with either of them, there is any interference with the circulation, the outlook is rendered worse.

The nature of the underlying disease is the most important factor dominating the course of the disease, and consequently influencing prognosis. As a complication of acute rheumatism, pericarditis must be regarded as an affection of no great severity, and the same may be said of those circumstances in which it accompanies one of the less serious general diseases. The case, however, is quite otherwise when pericarditis has its origin in some septic condition, or when it is associated with tubercle, cancer, or kidney disease.

The amount of fluid exudation plays an important part in the prognosis, and it may be laid down as an axiom that every additional ounce renders the case more serious.

The nature of the fluid which is present is of much importance, and instances of purely serous pericarditis are mild

in comparison with those in which pus makes its appearance. When the pus has become putrid, matters have reached a serious pass, and hæmorrhagic pericarditis has at all times to be regarded as one of the most grave conditions.

TREATMENT.—In the course of any affection likely to be complicated by pericarditis the question will naturally arise, whether it is possible to avert the tendency; in other words, whether prophylaxis may be advantageously employed. When salicin and the salicylates were first introduced in the treatment of acute rheumatism, and found to have such excellent results, it was fondly hoped that they would, in addition to cutting short the disease, obviate many of its complications. These hopes, unfortunately, have not been realised. It is impossible to speak with absolute certainty upon this point, because statistics, although collected by the most accurate observers, are open to misinterpretation. The relative frequency of pericarditis in acute rheumatism necessarily varies in some degree, and this consideration renders the comparison of statistics not absolutely reliable. When every aspect of the question has been considered, it must be admitted that pericarditis is probably as common under salicin treatment as under any other.

Many attempts have been made to prevent the onset of pericarditis in rheumatism and other diseases likely to produce it. Only two methods of procedure appear to have any influence. One of these consists in the systematic application of cold over the præcordia, either by means of the ice-bag, or iced cloths, or, still better, Leiter's tubes. The other consists in the continuous application of small blisters over the præcordia. This has been more particularly insisted upon by Caton, who speaks strongly in its favour.

If acute pericarditis has made its appearance, and is recognised by means of physical signs, the patient must be treated in accordance with general principles. It is sometimes unnecessary to adopt any special measures, and each case must be managed according to its particular features. The principal indications for treatment are to curb the process and remove its results. These indications may be fulfilled by diminishing the cardiac energy, while removing all painful and distressing

symptoms. The removal of the products of the disease may be aided by medical or surgical measures.

Absolute rest is necessary, and sleep is essential; if insomnia be a marked feature, it should be combated. Simple sedatives, such as the bromides, are, as a rule, quite sufficient for this purpose, but if there be much depression it is better to administer such a drug as paraldehyde, which will produce a stimulating effect in addition to producing sleep.

Milk diet is in most acute cases absolutely indicated, and if there be much thirst, abundant simple drinks may be given. After the first two or three days of the affection, different kinds of soup may be added to the dietary, which may be extended day by day in every direction.

The functions of the bowels must be watched, and, if there be constipation, an enema or saline aperient should be administered.

With regard to drugs, it need hardly be said that if pericarditis has arisen in the course of an acute general disease, the lines of treatment already adopted in that disease will require to be steadily pursued, and only in the case of definite indications is it necessary to administer special remedies, or to have recourse to further measures. In the case of rheumatic pericarditis, the combination of sodium salicylate with potassium bicarbonate gives most satisfactory results, from fifteen to twenty grains of each drug being administered every four hours.

Aconite is sometimes recommended, but is absolutely unworthy of confidence. There is only too great a tendency to reduction of cardiac energy as part of the disease, and drugs belonging to this class should be shunned. The salicylates are quite sufficiently depressing in themselves.

If the temperature, in spite of the action of the salicylate, tends to become too high, it is advisable to employ some antipyretic, and experience shows that the most useful drug for the purpose is phenacetin. It may be given while the salicylate is steadily continued. Antipyrin, antifebrin, and other remedies of the same class may, however, be used in some cases with advantage.

If the pulse has a tendency towards undue frequency, the administration of alcohol in any form most agreeable to the patient, while at the same time unlikely to interfere with the

functions of digestion, may be given, but it is often necessary at this point to step in with one of the cardiac tonics, such as digitalis or strophanthus.

If there be much pain, it may be relieved by the application of cold in any of the forms mentioned, but it is sometimes found that heat is more soothing to the patient than cold, and hot poultices or fomentations may in such instances be employed. In many cases it is very quickly relieved by the application of two or three leeches to the præcordia. When there is much distress along with great pain, general blood-letting, as advocated by Fagge, may be resorted to, and four or six ounces of blood removed from the arm. It must not, however, be forgotten that the pain may be so severe as to require the employment of morphine or some other derivative of opium. There is absolutely no danger in the use of these drugs, since opium not only regulates, but even increases, the cardiac energy when given in small doses. It may be applied externally by means of fomentations, or administered internally in any suitable form, but sometimes it is more advantageous to exhibit morphine hypodermically. This is more particularly the case when the digestive functions are disordered.

When effusion has taken place, new considerations arise. If the fluid remains moderate in amount, it is unnecessary to do more than maintain the treatment already employed, and to watch for the development of such symptoms as may be expected from the fluid exudation. If the fluid becomes large in quantity, diuretics and purgatives may be advisable, while counter-irritation is employed externally. It must be borne in mind that when the pericarditis is of renal origin iodine is to be applied instead of cantharides. The best remedies for internal use are iodide of potassium along with acetate of potassium.

On account of the danger produced by the intrapericardial pressure, the question of operative relief must always be borne in mind. Riolan proposed paracentesis of the pericardium, and this was also advocated by Senac. Desault performed paracentesis for a supposed pericardial effusion, but it was found on post-mortem examination that the case was one of circumscribed pleural effusion. The first actual paracentesis



of the pericardium was performed by Romero, who reported three successful cases to the Faculty of Medicine of Paris. The Faculty would not allow the report of these cases to be printed in their Transactions, since they declined to sanction the operation, but they are described by Mérat. Karawagen, Schuh, Kyber, and Aran were his immediate followers, and, like him, they were remarkably successful in their results. The most important writings regarding paracentesis are those of Trousseau, Roberts, Hindenlang, Clifford Allbutt, Grainger Stewart, Fiedler, and West. The statistics of all the known operations up to the year 1880 have been collected with indefatigable industry by Roberts, and his work is a storehouse of information upon the subject.

The earliest operations were performed by means of the bistoury and scissors, but during the intermediate period the trocar and canula were always employed. Since the introduction of the aspirator, it has for the most part been relied upon in the first instance, but in purulent or hæmorrhagic cases free incision has been practised.

The considerations which have to be borne in mind in connection with paracentesis pericardii are the determination of suitable cases, the selection of the most favourable site for operation, and the best method to be adopted. It is not difficult to determine when paracentesis pericardii is advisable. The presence of symptoms denoting great interference with the circulatory functions, and the physical signs of a considerable effusion into the sac, will furnish sufficient proof of the necessity or advisability of the operation. Opinions have differed a good deal as to the most suitable site for the operation, and, without discussing all the different views which have been advocated, it may be said that there are two positions which are least open to objection. The first of these is the fifth intercostal space just inside of the mammillary line; the second is the angle between the ensiform cartilage and the left costal margin. In most instances the latter is the most advisable site, inasmuch as by means of it the lowest portion of the pericardial sac can readily be reached, and with a distended sac there is but little likelihood of damaging any of the neighbouring viscera. Undoubtedly the

most suitable instrument for employment is the aspirator, by means of which fluid may be easily withdrawn without any risk of introducing air into the pericardial sac, but in those cases where the contents of the sac are purulent, it may afterwards be necessary to make a free incision with antiseptic precautions. Such an instance has recently been described by Underhill.

The great dangers attendant upon paracentesis are the possibility of wounding the lung or pleura in operating in the fifth intercostal space, or the superior epigastric artery if the site of the operation be in the angle between the xiphoid cartilage and costal margin, while in both localities there is a possibility of puncturing the heart. These risks are, however, probably somewhat exaggerated. If the fifth intercostal space be selected, the sac is as a rule so distended as to have pushed the left lung altogether aside, and the passage of the aspirator through the pleural sac is unattended by any trouble. If the epigastric site be selected, there is but little danger of wounding the superior epigastric artery if the aspirator be inserted quite close to the sternum, seeing that the artery is about half an inch away from the sternal margin. The third danger connected with the fear of puncturing the heart has also been greatly exaggerated, and such cases as those of Hulke, reported by Evans, and of Sloan show that even when the heart has been punctured and blood has been withdrawn from it accidentally, the symptoms are improved instead of rendered worse.

Roberts' statistics deal with sixty cases of paracentesis pericardii, twenty-four of which ended in recovery, while death occurred in the remaining thirty-six. The mortality, therefore, has been 60 per cent.; this is remarkably good considering the extremely serious nature of the affections for which the method was employed.

CASE 5. *Pericarditis from Rheumatism*.—F. F., aged seven, school-boy, was admitted to Ward 22 of the Royal Infirmary, 27th June 1893, complaining of breathlessness on exertion. His parents were both healthy, and all his brothers and sisters were perfectly well. Both his grandmothers were alive, and enjoyed good health. His paternal grandfather died of bronchitis; his maternal grandfather died of heart disease and

dropsy. These facts are mentioned in case the atavistic tendency manifested by the patient should perchance be of real interest. His social conditions had always been excellent. When he was eighteen months old he suffered from measles and whooping cough. Seven months before admission he had suffered from a sore throat accompanied by a rash, in regard to which no definite facts could be ascertained, and three months before admission he had passed through a severe attack of acute rheumatism from which he had never entirely recovered.

On examination the patient was found to be a bright intelligent boy, with a clear transparent skin through which a bright flush appeared high upon the cheeks. His temperature was normal. The tongue was almost clean, and there were no symptoms connected with the alimentary system. The pulse varied extremely in rate, fluctuating between 120 and 160 per minute. The vessel was empty and the blood pressure was low. The rhythm was regular and the pulsations were small.

On applying the hand to the præcordia, the apex beat was found to be diffuse, the impact of the heart was apparently behind the fourth rib, the right and left borders of the heart were respectively two and three inches from mid-sternum. On auscultation a loud but soft blowing systolic murmur was heard in the mitral area, and was propagated for about a couple of inches in every direction, but the most characteristic feature of the case was the presence of pericardial friction. This was heard over almost the entire præcordial region and extended slightly beyond it, both above and below. Over almost the whole of this area it was of twofold rhythm, systolic and diastolic, but in the region of the mitral and tricuspid areas it had a distinct threefold rhythm, presystolic, systolic, and diastolic. These appearances have been shown in Fig. 119, p. 331. One fact of much interest is that on auscultation through the dressing-gown and night-shirt only the systolic endocardial murmur could be heard.

The case was of interest especially on account of the fact that the triphasic rhythm was confined to the apical region, while it was diphasic at the base.

Under absolute rest and counter-irritation by means of some small blisters, followed by moderate doses of digitalis along with iron, the patient rapidly improved and was discharged free of all pericardial symptoms in the course of three weeks.

CASE 6. *Rheumatic Endocarditis followed by Pericarditis.*—J. C., aged 20, soldier, was placed under my care in Ward 22 of the Royal Infirmary by Surgeon-Captain Will, on 5th August 1892, complaining of pains in his joints and breathlessness. No special hereditary tendencies could be elicited, and apart from the inevitable exposure connected with garrison duty, there was nothing in his social conditions predisposing to



disease. He had been perfectly healthy all his life. Three days before admission he began to suffer from pains in his joints and feverishness.

The patient's temperature on admission was a little over  $103^{\circ}$ . The face was pale, and the skin was bathed in profuse perspiration, with characteristic rheumatic odour. The tongue was covered by a white fur, but there were no symptoms connected with the alimentary system. On admission there were no symptoms indicating any cardiac implication. The pulse rate was a little over 100. The fulness was slightly below, while the compressibility was slightly above the normal, and the pulse was perfectly regular. The only point, on examination of the præcordia, calling for mention is that the right border of the heart was slightly enlarged, extending two inches and a half from mid-sternum. During the few days which elapsed after the patient's admission it was obvious that endocardial changes were in progress. The heart was not merely enlarging, but, in addition to systolic murmurs in the mitral and tricuspid area, undoubtedly produced by dilatation, there was an aortic

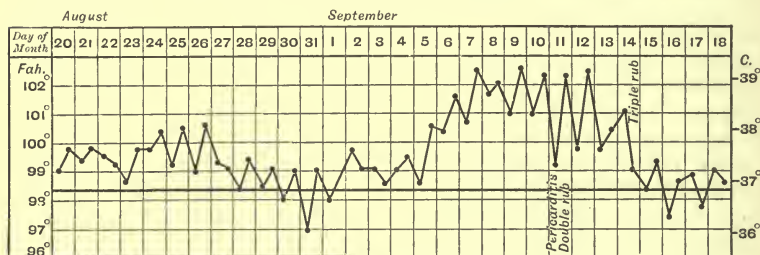


FIG. 120.—Temperature chart from Case 6.

systolic murmur. By the 15th August such suspicions were absolutely confirmed. The pulse, still frequent but perfectly regular, had become empty and compressible, and manifested well-marked diastolic murmur; on elevating the arm, moreover, there was considerable tendency towards the water-hammer pulse. Inspection of the præcordia gave no abnormal symptoms, but on palpation the right border of the heart was found to be three, and the left four and a half inches from mid-sternum. On auscultation there were loud but soft blowing systolic murmurs in the mitral and tricuspid area. There was a distinct aortic systolic murmur, and occasionally a soft diastolic murmur could also be heard. The second sound in the pulmonary area was considerably accentuated. At this period, therefore, it was clear that along with dilatation of the heart, produced by the pyrexia, there was endocarditis affecting the aortic cusps. The temperature, which under the influence of salol had fallen at once, was seldom above  $100^{\circ}$ , yet it never quite fell to the normal; by the end of August, however, it was only  $99^{\circ}$  in the evenings, and subnormal in the mornings.

On 5th September the temperature began again to rise, and on the 7th it almost reached  $103^{\circ}$ . The pulse also increased in frequency, yet the most sedulous examination of the heart failed to reveal any cause. On the 11th September intense præcordial pain began in the forenoon,



and early in the afternoon a distinct pericardial friction sound was present. It was characterised by a distinct double rhythm, systolic and diastolic. During the next two days the condition remained unchanged, except for a fall in the temperature to little above  $100^{\circ}$ ; on the 14th September, however, at the base of the heart there was a most distinct triple rhythm in the friction sound.

The temperature from this date rapidly came within ordinary limits, and the friction speedily waned. Little or no serous effusion took place, for the area of cardiac dulness became smaller, and the patient was dismissed a couple of weeks afterwards in a fair state of general health. The tricuspid systolic murmur had disappeared, the mitral systolic murmur was shorter and sharper, and the accentuation in the pulmonary area had disappeared. There was, however, a loud rough systolic murmur, followed by a soft diastolic murmur, in the aortic area.

This case is interesting as showing a contrast to Case 5 as regards the localisation of the triphasic friction sound.

CASE 7. *Pericarditis from Bright's Disease.* — T. W., aged 35, engineer, was admitted to Ward 22 of the Royal Infirmary, 28th June 1893, complaining of shortness of breath and cough. His family had no special disease tendencies, and his social conditions had always been comfortable, but his occupation exposed him to sudden vicissitudes of temperature. His previous health, nevertheless, had been perfectly good. Two months previous to admission he had a severe chill when fishing, and he had never been well since that time. On examination the patient manifested a considerable degree of hebetude. In answer to questions his utterance was extremely slow, although quite distinct. There were some muscular twitchings in the face as well as in the extremities. The temperature was  $97^{\circ}$ .

The lips and mouth were extremely dry, the tongue of a bright red hue with a glazed surface, and the teeth much decayed. There was frequent vomiting, and there had been constipation. None of the abdominal viscera showed any abnormality. The patient complained of pain below the left mammilla, which was increased with every movement and distinctly aggravated by percussion. The pulse was about 100 per minute. The vessel was full and the pressure high. The pulsation was perfectly regular and distinctly tardy. The apex beat was in the fifth left intercostal space, four inches from mid-sternum. The impulse was forcible and prolonged. The right and left edges of the heart were respectively two and a half, and four and a half, inches from mid-sternum, and on auscultation there were distinct mitral and tricuspid systolic murmurs. The second sound was nearly equal in intensity in the aortic and pulmonary areas, and was in both distinctly accentuated. There was a copious frothy expectoration stained with blood. At the bases of both lungs the percussion sound was somewhat muffled, the breathing tended towards the bronchial type, and was accompanied by crepitations. There was considerable pain in the loins. The urine amounted to forty ounces.

It was pale in colour and acid in reaction, with a specific gravity of 1013. It contained 187 grains of urea and a large amount of albumin. Hyaline and granular casts were present. In addition to the obvious nervous phenomena above referred to, the patient suffered much from headaches, and was sleepless. It was clear that chronic nephritis had been left by a previous acute attack, and that it was accompanied by dilatation and hypertrophy of the heart. The patient was treated by means of milk diet, along with vapour baths and purgatives, while

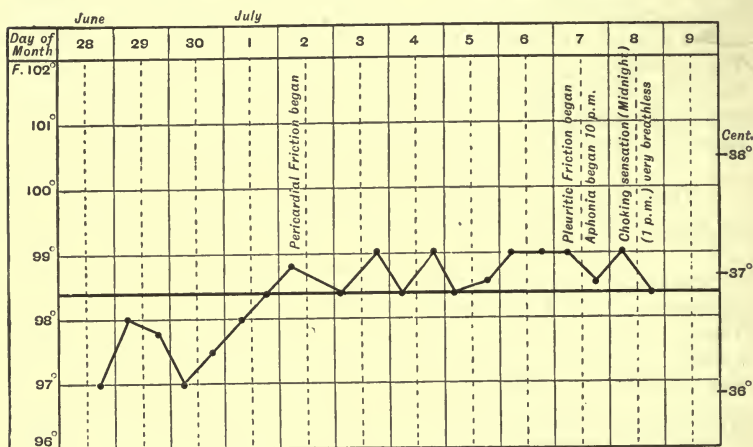


FIG. 121.—Temperature chart from Case 7.

sulphonah was administered in order to ensure sleep. On 30th June, a diuretic mixture was begun.

On 2nd July, auscultation revealed the presence of a to-and-fro friction murmur, which was most intense towards the base of the heart. Three leeches were applied to the præcordia. During the next four days little change occurred in the patient's condition, but on the 7th July pleuritic friction made its appearance over the right side, and, at the same time, comparative dulness was found on percussion over the base of the left lung, which was accompanied by great dyspnœa, and became almost absolute, while the breath sounds and vocal resonance disappeared, showing the presence of hydrothorax. The following day the breathlessness became excessive and aspiration had to be resorted to, but in spite of every endeavour the patient died. No autopsy was obtained.

**CASE 8. Pericarditis from Pleuro-pneumonia.**—L. G., aged 21, domestic servant, was admitted to Ward 25 of the Royal Infirmary, 15th December 1895, complaining of cough and pain in the chest.

Her family history was satisfactory, and her surroundings had always been good; her previous history had also been satisfactory. The illness for which she was admitted began two days before with a well-marked rigor, attended by severe pain in the left side of the chest and a harsh, hacking

cough. On admission, the patient was found to be well-nourished and muscular. Her face was pale, with a brilliant flush upon each malar prominence. Her temperature was  $103^{\circ}$ ; the pulse and breathing 126 and 44 per minute. There was a copious expectoration of rusty-coloured sputum, which, it may be mentioned, on further investigation, was found to contain numerous pneumococci. The tongue was coated with a brown fur and was somewhat tremulous. The digestive system otherwise gave no indications, and there were no symptoms connected with the circulatory organs. On examination of the chest, it was observed that the left

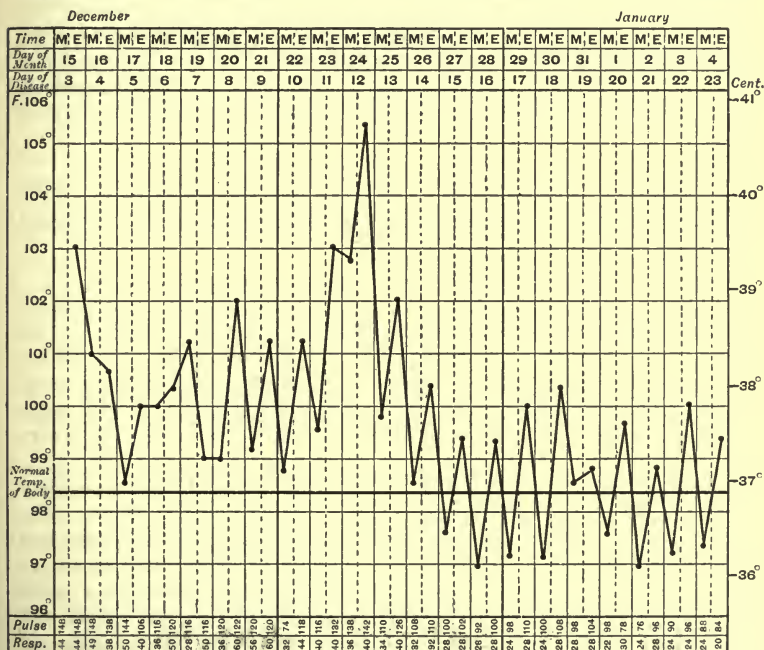


FIG. 122.—Temperature chart from Case 8.

side did not move so freely as the right. The vocal fremitus was increased over the lower portion of the left lung. On percussion there was almost absolute dulness over that area, and on auscultation there was high-pitched bronchial breathing, with a few fine crepitations on inspiration, and greatly increased vocal resonance. The urine contained almost no chlorides, and had a very copious precipitate of urates. The patient was in a somewhat soporose condition with slight muttering delirium at night. The case was obviously one of pneumonia, and from the considerable pain present, as well as from the friction sound, it was clear that a certain amount of pleurisy was also present. During the following day the temperature came down somewhat sooner than had been expected, but on the following day or two it oscillated considerably.



On the 19th it rose still higher. On this date examination of the heart, which had been sedulously carried out day by day, revealed the presence of distinct pericardial friction accompanying the systole and diastole of the heart. This friction sound was heard over almost the entire præcordia, and it was unattended by any symptoms. In two days it entirely disappeared, but, with its disappearance, an ominous flush upon the *alæ nasi* and adjacent portions of the cheeks made its appearance, and the temperature began again to run up. A day later there could be no mistake as to the condition which was present, for the flush was obviously erysipelatous, and the temperature was still rising. The patient was therefore transferred to the observation wards and carefully tended there. In five days, under treatment with sulpho-carbolate of sodium, the erysipelas had disappeared, and when all danger of infection had been removed, she was again transferred to Ward 25, where her recovery went on rapidly.

This case could only be regarded as an instance of pericardial infection by means of the pneumococcus, as the erysipelas distinctly followed the pericarditis.

CASE 9. *Septic Pericarditis*.—C. P., aged 75, retired manufacturer, began to complain in April 1893 of a swelling above the clavicle on the right side, along with a dull pain in the chest and weakness. The family tendencies presented no facts bearing upon his case, and his social conditions had always been excellent. He had during his earlier years suffered from small-pox and from typhus fever, and he had met with several accidents.

The swelling of the neck began insidiously, and it had been preceded by a hard, dry cough which had existed for some weeks before. The patient's appearance was somewhat sallow, although his complexion had previously been good. The skin was constantly moist, the temperature rose a couple of degrees at night, the tongue was covered by a moist yellow fur; the pulse ranged between 120 and 130—it was empty and compressible, with a regular but small pulse wave. There were no symptoms either on inspection or palpation of the præcordia, and, on percussion, the upper level of cardiac dulness was at the third left costal cartilage, the right border two inches and a half to the right, and the left three inches to the left of the sternum. Examination of the respiratory system failed to reveal any cause for the cough. The condition of the urine was absolutely normal. The skin, as already mentioned, was constantly moist, and on the least exertion profuse perspiration was induced. Sleep was disturbed, but the patient suffered from no headache or other over-nervous symptom.

In this case there were few definite characters. The evening rise of temperature and the condition of the skin caused a suspicion that septic infection might be the origin of the condition, and this became more evident in the course of two or three days, when the cervical glands assumed a softer consistence, and fluctuation was elicited in them. Mr.



Joseph Bell saw the patient along with me and incised the suppurating glands, while quinine was administered in large doses. The surgical intervention produced no amelioration in the symptoms. The temperature continued to rise at night, and the profuse perspirations occurred as before, while the pulse rate remained steadily between 120 and 130. The pain in the chest also tended to become worse instead of lessening, and the feeling of weakness also increased. It was obvious that the pulse rate was kept up either by some general toxic agent, or by some local disturbance. The most careful examination of the circulatory organs gave no symptoms further than those which have been referred to, and it could only be a matter of inference as to the nature of the irritation keeping up the pulse rate. We feared the development of some deeply-seated suppuration connected with the mediastinal glands, possibly involving the pericardium, but of this there was absolutely no proof. On consultation with Mr. Joseph Bell, it was arranged that the patient should seek the benefit of change of air, and he therefore went to the country, and was placed under the care of Dr. Hall, of Rothesay. After remaining in the country for two or three weeks, he returned in May to Edinburgh with his condition practically unchanged. His temperature still rose at night, his pulse maintained the same frequency of rate, the profuse perspirations constantly recurred, and the objective symptoms remained as they were before. Early in June the condition of the patient changed for the worse. He began to emaciate rapidly, and the weakness, always present, increased rapidly. The temperature, which had been always moderate, now became somewhat higher at night, and the perspirations became more excessive. The heart sounds enfeebled steadily and the pulse rose to 140 and 150, but on most careful examination of the præcordia, no change could be determined. The downhill course of events swiftly brought about a fatal termination on the 20th June. A post-mortem examination was performed the following day by Dr. Leith, which, omitting unnecessary details, revealed the fact of septic suppuration of the posterior mediastinal glands with implication of the pericardium, which contained a quantity of purulent fluid, abounding in streptococci.

This case furnishes an excellent instance of septic pericarditis, in which the circulatory complication although suspected could not be diagnosed with confidence.

### ADHERENT PERICARDIUM.

Amongst ancient writers obliteration of the pericardial sac was regarded as a congenital absence of the membrane; Vieussens and Lancisi are the first authors who recognised the true character of the condition. Senac, Morgagni, and other observers gradually unfolded many of the clinical

as well as anatomical features of the condition, but it is to Sibson, Friedreich, and Reigel that we are indebted for the most careful study of the symptoms of the disease.

**ETIOLOGY.**—It appears probable that synechia of the pericardium may follow in some degree every variety of pericarditis, but it is undoubtedly much more common in certain types. Fibrinous pericarditis is more likely to give rise to adhesions than any of the other forms, with the exception of the pericarditis which is an accompaniment of renal disease. In this form, as already mentioned, partial or general pericardial adhesions are frequent. The formation of adhesions may occur in an early stage of the disease, while a perfectly dry fibrinous pericarditis is present, or it may, and much more commonly does, take place after the reabsorption of an effusion. Possibly feeble action of the heart may tend to its production, and increased energy may cause the adhesions to break away. It appears to be a common result in those forms of pericarditis which become chronic, and more particularly in those which recur, a good example of which will be described below. Weakening of the energy of the heart must certainly act as a predisposing agent. It is an extremely common experience to find pleural as well as pericardial adhesions, and synechia pericardii is in many instances found associated with other pulmonary affections.

Statistics have often been collected with regard to the frequency of adherent pericardium, and vary very widely. Leudet, for instance, from the investigation of 1003 necroscopies, states that partial occur in 5 per cent., and total adhesions in 2·5 per cent. of autopsies, while the results of the Charité Hospital of Berlin, according to Breitung, give 156 cases of adherent pericardium in 324 cases of pericarditis.

**MORBID ANATOMY.**—The extent of the adhesions is extremely variable—from the slightest attachments by means of a few fibrinous bands to complete synechia there is an unbroken series of intermediate forms. The union is effected by layers of fibrin in which a certain number of cells are entangled. The appearance on section is shown in Fig. 123. The adhesions may take the form of bands or mats, which are mostly found at the base. Further changes occur, of which the

most common is fatty degeneration of the newly-formed tissue, although calcareous infiltration not infrequently occurs.

According to Sibson, when the adhesions are long and loose, and the heart is free from valvular disease or other influence tending to produce hypertrophy, the size of the heart is usually unaltered. If, however, the adhesions are short and powerful, and there are any pleuro-pericardial attachments, there is a greater tendency to hypertrophy. According to this observer, in two-thirds of his cases without valvular disease (12 in 19) the heart was considerably enlarged; in one-fifth of them (5 in 19) it was rather large; and only in one-tenth of them (2 in 19) the heart was of normal size. When peri-

cardial adhesions are attended by valvular disease, as is usually the case, the heart is invariably enlarged. The enlargement is usually due to combined dilatation and hypertrophy, and there is a great tendency in all cases to degeneration.

**SYMPTOMATOLOGY.**—Cases of adherent pericardium, latent throughout their whole course, may be discovered accidentally by physical examination, or only, indeed, revealed after death. On the other hand, there may be such symptoms as palpitation, breathlessness, cyanosis, and œdema. Ascites occasionally presents itself, in which the default of all possible causes, with the exception of some history of a pericardial lesion, raises the suspicion that adhesions, by compressing the inferior vena cava, are the source of the affection. In a few instances of adherent pericardium anginous symptoms make

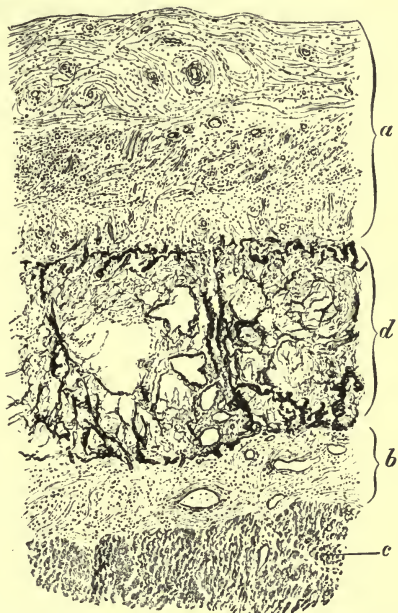


FIG. 123.—Fibrinous pericarditis ( $\times 20$ ) showing :  
a, pericardium ; b, epicardium ; c, myocardium ;  
d, fibrinous exudation showing organisation  
from both serous surfaces.



their appearance, and towards the end of life syncopal attacks are prone to occur.

The pulse sometimes presents no abnormal features. It often, however, is irregular, and is marked by greater oscillations of pressure than are present in health. There is frequently a well-marked dicrotic tendency. Not infrequently the paradoxical characters described by Kussmaul in mediastino-pericarditis are present, as was the case in a well-marked instance described below.

On examining the chest, there is occasionally to be seen a fulness in the left part of the præcordia, but more common than this is a flattening of the chest wall to the right, as well as to the left, of the region of the apex beat. When the movements of the heart are carefully inspected in patients who are reasonably thin—and in my experience adherent pericardium is mostly found in those who are slender—it will be seen that there is a retraction of the left half of the epigastrium with each beat of the heart, a symptom which was described by Kreysig, whose attention was called to it by Heim. There is also a drawing in of the intercostal spaces surrounding the apex beat, to which Riegel has devoted much attention, and in the lateral as well as posterior part of the chest, as insisted on by the younger Broadbent. Skoda held that the apex beat was drawn in with each systole, and this has been more particularly insisted upon by Friedreich and Guttmann. These statements are due to faulty observation, and are also based upon misconception of physiological facts. The retraction of the epigastrium, and of the interspaces surrounding the apex beat, may be determined in almost every case of adherent pericardium, but there is no indrawing at the apex itself. It has already been shown, from the physiological point of view, that, while during systole the ventricular portion of the heart lessens its transverse and antero-posterior diameters, it does not diminish its long axis, and this explains how there is retraction around, but not at the apex. Inspection also shows another weighty symptom, that inspiration is not accompanied by the normal expansion of the inferior part of the left side. If the patient be caused to change his posture, it will be found that it does not move with change of



position, that, in short, it retains the same position whether the patient lies upon his right or left side. As was observed by Sibson, the inspiratory movements of the central portion of the abdomen are considerably diminished. The central movements in health are from two to three times as great as the lateral movements of the abdomen, but in adherent pericardium the amount of movement is in some cases about equal in the central and lateral portions. It is by no means uncommon to find that during inspiration there is an indrawing of the lower portion of the sternum. An interesting fact, originally observed by Friedreich, is that during the diastole of the heart there is a most distinct collapse of the veins of the neck. One very important symptom is the diastolic rebound of the heart. This appearance, which can be appreciated by inspection, but more thoroughly realised by palpation, is one of the most characteristic features of the disease. On applying the hand, there is said to be weakening of the apex beat, unless it is accompanied by hypertrophy of the heart. No case of simple adherent pericardium has ever presented itself before me. There has been some valvular affection in every one, accompanied by a considerable amount of hypertrophy. In the majority of cases of adherent pericardium, as has already been shown, there is more or less hypertrophy, and the apex beat must in these be increased in force. The area of cardiac dulness entirely depends upon the presence or absence of hypertrophy or dilatation. As a rule it is enlarged, both to right and left, and this is more particularly seen when pericardial obliteration is accompanied by valvular lesions. The dulness does not change its position on any alteration of the posture of the patient. It also undergoes no modification on the deepest inspiration and expiration.

On auscultation the heart sounds are sometimes weakened, but on the other hand, in cases where there is hypertrophy, they are distinctly accentuated. It is impossible for me to say anything definitely on this point, seeing that no uncomplicated case has afforded me an opportunity for its observation. A metallic character of the heart sounds has been described by Riess.

DIAGNOSIS.—The recognition of obliteration of the pericardial sac is a matter of careful physical exploration, so frequently is it latent and absolutely unattended by any general symptoms. No single physical sign can be regarded as of much diagnostic value, with the sole exception of the immobility of the heart in varying postures, and the diastolic rebound of the parietes. The systolic retraction of the epigastrium and of the intercostal spaces around the apex beat may occur in any condition in which there is cardiac hypertrophy along with lack of elasticity in the lungs. The diastolic collapse of the cervical veins is of some utility when added to the other physical signs, but in itself it cannot be regarded as in any degree pathognomonic.

The diagnosis, therefore, of *synechia pericardii* must be based upon the assemblage of symptoms present; and if there be a paradoxical character of the pulse, a diastolic collapse of the cervical veins, a systolic retraction of the epigastrium and intercostal spaces, a fixed position of the apex beat under all conditions, an immobility of the area of cardiac dulness, and a diastolic rebound of the chest wall, the diagnosis of adherent pericardium may be regarded as certain. The characters of the heart sounds are too variable, and bear too little relation to the condition of the pericardium, to be of any value. It need hardly be added that if there is a history of previous pericarditis, it will render the diagnosis more certain.

PROGNOSIS.—The outlook in obliteration of the pericardial sac in itself is in no respect serious, inasmuch as the heart is surrounded by pulmonary tissue marked by such a high degree of extensibility and elasticity; the mere fact that there are adhesions between the pericardium and epicardium cannot in itself seriously interfere with the movements of the heart, and, as has been seen, uncomplicated adherent pericardium does not produce any great tendency to hypertrophy. When obliteration of the pericardial sac, however, is associated with valvular lesions, the complex pathological condition is one of most serious import. Not only does the heart suffer from the disturbances produced by the valvular lesions, but the additional strain, which it undergoes in consequence of the external adhesions, gives rise to a greater tendency to hypertrophy, and the heart,

as will be explained more fully in a subsequent section, is prone to grow beyond its nutritive possibilities. When there is an association of valvular lesions and pericardial adhesions, there is a great liability to heart failure, and some of the most striking instances of sudden death take place under such circumstances.

**TREATMENT.**—During the resolution of pericarditis it would seem probable that prophylactic measures might aid natural processes and lessen any tendency to pericardial adhesions. The use during this stage of deobstruent remedies, as iodide of potassium, together with external counter-irritation, would seem on theoretical grounds to be advisable; and if, during the period of regression, the heart in any case should give evidence of weakness, it would certainly seem advisable to use such remedies as are calculated to increase its energy. The combination, therefore, of digitalis with iodide of potassium is to be strongly recommended.

When adhesions have formed, medical measures are powerless, and treatment can only be symptomatic. In simple cases of pericardial obliteration there is but little interference with circulatory processes, and all that is necessary in such cases is to recommend abstinence from all excessive physical exertion, which might at once put a heavy strain upon the heart, and cause considerable oscillations of intrapulmonary pressure. The food, at the same time, ought to be such as will not cause any tendency to gastric dilatation. When, in consequence of adherent pericardium, there is a tendency to cardiac failure, its symptoms must be met by the method which will be afterwards fully described for cases of that description.

**CASE 10. *Adherent Pericardium.***—F. B., aged 24, stationer, was under my care in Ward 22 of the Royal Infirmary, complaining of uneasiness in the chest and breathlessness on exertion. The patient's family seemed entirely free from all hereditary tendencies connected with his affection, as his father had died at the age of sixty of a tumour in the neck, and one sister had died from a suppurating affection of her arm, while the patient's mother, two brothers, and four sisters were in the enjoyment of perfect health. His environment had been all that could be desired. The patient suffered at the age of twelve from a severe attack of acute rheumatism, which was repeated at the age of sixteen, and again



when he was seventeen years old. During the seven years which had elapsed between the third attack of acute rheumatism and the present illness the patient had felt perfectly well. Four months before admission the attack for which he came under treatment began. There was at first merely a feeling of oppression and of breathlessness on any unwonted exertion or excitement; this had become more pronounced, so that there was a constant feeling of uneasiness and weight in the chest, along with breathlessness on the least exertion.

The patient complained of no symptoms beyond those which have been mentioned. His digestive functions were absolutely normal in every respect. His teeth were by no means good, but the tongue was clean. The patient was fair with a clear skin, through which the blood mantled brightly upon the malar prominences. The lips were ruddy, and the conjunctivæ well coloured. The tint of the nails was bright, and there was no arching of them or clubbing of the fingers. There were no abnormal symptoms referable to the hæmopoietic viscera. The pulse was of extremely variable frequency, the rate oscillating between 70 and 90. The vessel was somewhat empty and the blood pressure was low,

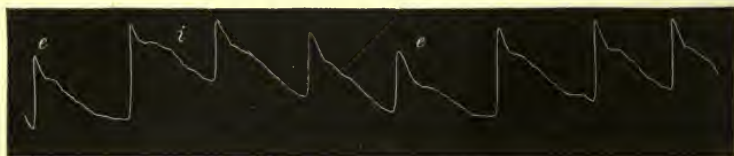


FIG. 124.—*Pulsus paradoxus* in adherent pericardium; pressure 3 oz.

but each wave was high, although collapsing. It presented, in short, some of the features of Corrigan's pulse. The fact in regard to it, however, which was of greatest interest, was connected with its relation to respiration. Careful investigation by means of the finger revealed the fact that during deep inspiration the pulse wave became smaller, while it again during expiration increased in volume. The pulse furnished, in short, as far as could be judged by the finger, the characters usually termed paradoxical. A tracing obtained with Marey's sphygmograph brought out these characters very distinctly, and they are shown in the accompanying illustration (Fig. 124).

On examining the patient's neck and chest, two appearances at once caught the eye. There was a considerable increase in the arterial impulse in the neck, but this was entirely overshadowed by the phenomena seen in the lower part of the præcordia. The systole of the heart, shown by the bounding arteries of the neck and a pronounced apex beat in the sixth intercostal space, was accompanied by a sinking in of the left half of the epigastrium, in the angle between the ensiform cartilage and costal margin, and by a most distinct recession of the fourth, fifth, and sixth intercostal spaces, both inside and outside of the apex beat, which was four inches and a half from mid-sternum. The systole was succeeded by a very well marked diastolic rebound, which could be seen over the



entire præcordia. On applying the hand, the apex beat was felt to be violent, and it gave the hand a distinct impact. All the parts, however, around the immediate apex beat withdrew themselves from the hand, and this could also be made out very distinctly in the epigastrium. Far from there being any recession in this case of the apex beat, therefore, there was a most distinct, almost violent, impulse. Tracings were obtained by means of the cardiograph from the apex, which is reproduced in Fig. 125.

On percussion the cardiac dulness was found to begin at the third costal cartilage on the left side. The right margin at the level of the fourth rib was two inches and a half, while the left was five inches from mid-sternum. At the level of the xiphoid cartilage, however, the left margin of the heart was five inches and a half from mid-sternum. The heart was therefore greatly hypertrophied. On auscultation a shrill, rough, systolic murmur was heard, with its maximum intensity in the mitral area, propagated for a considerable distance in every direction. The first sound in the tricuspid area was distinct, although



FIG. 125.—Cardiogram from a case of adherent pericardium.

accompanied by the mitral murmur. In the aortic area systolic and diastolic murmurs were heard, of which the former had its maximum intensity half-way up the manubrium sterni, and was propagated into the carotid and subclavian arteries, while the diastolic murmur was most distinctly heard about the level of the fourth costal cartilages. In the pulmonary area there was an accentuated second sound. It was evident that in this case there was obstruction as well as incompetence at the aortic orifice, and from the high-pitched and harsh characters of the mitral murmur, it could not be doubted that there were also obstruction and regurgitation at the mitral orifice. There were no symptoms calling for remark connected with any of the other systems, and they may be passed over without comment. Absolute rest followed by some resistance exercises after a few days relieved the patient of all his troublesome symptoms, and he was discharged much improved.

The patient furnished an excellent example of the physical signs of adherent pericardium, and it is of interest to note the salient features of the case. There was an entire absence of any diastolic collapse of the cervical veins. A distinct impulse was produced by the apex, accompanied by a recession of all

the surrounding parietes, and followed by a very pronounced diastolic rebound. The paradoxical pulse is of especial interest as showing the worthlessness of this appearance in the diagnosis of mediastino-pericarditis.

### MEDIASTINO-PERICARDITIS.

During the course of pericarditis, as was previously mentioned, there is occasionally a tendency for the morbid processes to pass beyond the pericardium, and to involve the textures of the mediastinum. As a consequence of this extension of the affection there is at times a considerable amount of thickening of the mediastinal tissues with the formation of adhesions of different kinds, which may implicate the great vessels in the neighbourhood of the base of the heart. Griesinger, according to Widenmann, about forty years ago, had occasion to observe a patient in whom a purulent pericarditis was associated with fibrinous mediastinitis. In this case a considerable amount of induration of the connective tissues surrounding the great vessels was found on post-mortem examination, and the newly formed tissue had produced a considerable amount of constriction of the venous trunks, as well as of the aorta. There was almost entire obliteration of the pericardial sac, except at one point, where a circumscribed space containing pus was seen. It was observed during life that the pulse showed a distinct intermission during inspiration. About twenty years later Kussmaul observed similar cases in which he suspected an indurated mediastino-pericarditis on account of the state of the pulse. He was enabled, therefore, to study the conditions presented by such cases.

**ETIOLOGY.**—It is probable that the ordinary causes of pericarditis may, under conditions favouring their development, produce such extension of the morbid processes as to involve the mediastinal textures; but in some of the cases which have been described the lesions have had their origin in affections of the lungs, pleuræ, or mediastinal glands.

**MORBID ANATOMY.**—Several different pericardial lesions have been described under indurated mediastino-pericarditis. Fibrinous, serous, purulent, or hæmorrhagic pericarditis, with

adhesions, as well as hypertrophy, dilatation, or degeneration of the myocardium, have also been seen. Various lesions of the lungs and pleuræ were present in some of the cases which have been recorded, but an essential feature of this particular affection is the presence of membranous or cord-like adhesions, directly or indirectly binding the base of the heart and the great blood vessels to the mediastinal textures, to the sternum, to the spinal column, or to the œsophagus.

**SYMPTOMS.**—In some of the patients whose symptoms have been carefully recorded, breathlessness, cyanosis, and dropsy have been present. The principal features of the affection, however, are derived from careful physical examination.

On examination of the pulse it has, as a rule, been found to be somewhat empty, of moderate pressure, and considerable frequency, but its principal character is a fall of pressure, or a degree of intermission during inspiration; that is to say, the pulse presents the characters of the *pulsus inspiratione intermittens*, or *pulsus paradoxus*. On inspecting the neck, the jugular bulbs, or even the entire visible veins of the neck, are described as swelling up during every forced inspiration and subsiding again on expiration. The *præcordia* has sometimes presented some appearances connected more or less directly with the condition. There has been, for example, inspiratory retraction of the epigastrium, as well as systolic retraction of the intercostal spaces surrounding the apex beat. Palpation has shown that the apex beat remained fixed in every posture which the patient assumed, while percussion has also shown some enlargement of the cardiac dulness. On auscultation the heart sounds have sometimes been unaltered, but they have also been described as weak and muffled. The important point is that there has been but little change during inspiration and expiration, notwithstanding the change in the pulse.

**DIAGNOSIS.**—The recognition of this particular form of pericardial affection must be admitted to rest upon a somewhat uncertain foundation. The two features which have been relied upon in establishing the diagnosis are connected with the arterial pulse and the cervical veins. Traube, Bäumlér, and Gräffner observed the paradoxical pulse in cases



of serous pericarditis without any affection of the mediastinum, and later observers have frequently substantiated their observations. The same appearances have been observed in aneurysm, and Riegel has discovered that the pulse may give characters extremely like, if not absolutely identical with, those of the *pulsus paradoxus* in individuals apparently healthy. It is therefore clear that the paradoxical pulse in itself cannot be relied upon as an infallible proof of adhesions surrounding the great blood vessels. The inspiratory swelling of the veins of the neck must, however, be allowed rather more weight when taken together with other symptoms proving the implication of the pericardium.

PROGNOSIS.—The outlook in cases of this nature will depend entirely upon the attendant conditions, since the fact of adhesions about the base of the heart and the great vessels, although interfering considerably with some of the circulatory functions, does not necessarily involve serious consequences.

TREATMENT.—The management of any case in which mediastino-pericarditis is present must be regarded as entirely symptomatic. If pain be a prominent symptom, it may in most cases be speedily removed by the application of two or three leeches to the *præcordia*; but in some instances their application is not sufficient to procure relief, and the subcutaneous injection of morphine may be required. If the temperature tends to become too high, the employment of phenacetin, or its addition to such remedies as have already been employed, may prove useful, but the local application of the ice-bag, or of Leiter's tubes, must also be taken into consideration. When frequency and irregularity of the pulse make their appearance, it may be necessary to administer diffusible stimulants, such as alcohol and ammonia. It may further be absolutely necessary to prescribe *digitalis* or *strophanthus* for cardiac failure.

### PNEUMOPERICARDIUM.

The existence of gas in the pericardial sac was observed by Voigtel and briefly described by Laennec. The most important observations upon the subject which have since



been made are those of Graves, Bricheteau, Stokes, Friedreich, and Begbie. Pneumopericardium is without doubt one of the rarest of cardiac affections. Schrötter states that, during thirty-five years' service amongst the enormous clinical material of the General Hospital of Vienna, he has never had the opportunity of seeing this affection, and Skoda had a similar experience. A very large proportion of hospital physicians have the same tale to tell.

**ETIOLOGY.**—Pneumopericardium owes its origin to causes falling into two groups, which may be termed fistulous and traumatic. Produced by the first class of agents are those cases in which, from extension of morbid processes in adjacent structures, a communication is established with air-containing viscera. Illustrations of this are to be found in extension from pyopneumothorax, as described by Eisenlohr; from a pulmonary cavity, by Stokes; from cancer of the œsophagus, by Begbie; from gastric ulcer perforating the diaphragm, by Säxinger; and from hepatic abscess perforating both stomach and pericardium, by Graves. Under the second category fall those cases in which the entrance of air into the pericardial sac is due to injury. Openings into the pericardial sac through the chest-wall have been described by several authors—gunshot wound, by Bodenheimer; incision by sharp instruments, by Feine; puncture by a trocar, by Aran; fracture of the ribs, by Morel-Lavallée. A similar cause has also been described by Walshe, in a most interesting case, where an oriental juggler perforated the pericardium in the attempt to swallow a long blunt instrument.

The belief that gas may be spontaneously developed within the pericardial sac has never been altogether left without witness. A number of instances have been recorded in which such an occurrence was accepted as the cause of pneumopericardium, and one of the most recent writers upon the subject, Petit, confidently asserts that an event of the kind is only a particular instance of the phenomenon, very well known in the present time, of pneumatosis taking place in closed collections of pus. He even states that this cause appears to be tolerably frequent. It cannot be denied that on post-mortem examination, gas is occasionally

found in the pericardial sac. The patients have, for the most part, during life manifested no clinical features connected with the pericardium, and on investigating such cases, they are almost always found to have been instances in which there was a considerable amount of post-mortem decomposition with all its possibilities. Such instances cannot therefore be accepted as proof of a spontaneous generation of gas within the pericardial sac. The statements with regard to the presence of gas within closed collections of pus are open to question, not in respect of fact, but of theory. It is to be borne in mind that cases of abscess containing gas have always been found near some viscus in which air is present, or in superficial parts to which ready access from the atmosphere has been permitted. The inference is obvious. It is equally clear that a small communication established between the pericardial sac and one of the air-containing organs surrounding it may rapidly heal, the air being closed within the sac and gradually absorbed by its membrane. Such is the probable explanation of the interesting cases described by Love and by Lundie. The analogy of pneumothorax renders this suggestion more than likely. Cases of what have been called spontaneous pneumothorax, *i.e.* the entrance of air into the pleural sac without any known previous disease, are seen occasionally. An instance was recently described by me, to which was appended a summary of similar examples previously recorded. In such cases, after the air has obtained admission the wound heals up and the air is gradually absorbed. There is no difficulty in applying similar reasoning to the case of the pericardium. It must further be remembered that many at least of those cases in which spontaneous pneumopericardium has been believed to be present, and in which the patient has recovered with astounding rapidity, afford considerable possibilities of error in diagnosis. It probably falls to the lot of most hospital physicians to be asked to see patients in whom pneumopericardium has been diagnosed upon insufficient evidence—cases in which dilatation of the stomach has caused some of the clinical features of pneumopericardium.

The discovery, a few years ago, of the bacillus *aërogenes capsulatus* by Welch, and its further study by him in associa-

tion with Nuttall and Flexner, have gone a long way to clear up some difficulties surrounding the subject. The organism has been found to be the cause of subcutaneous emphysema in connection with wounds, and when this has occurred, gas is found in the blood and tissues generally, as well as in the pericardium, pleura, and peritoneum. In traumatic cases, therefore, pneumopericardium is possible.

MORBID ANATOMY.—Pneumopericardium has invariably been attended by pericarditis, and pathological anatomy has therefore revealed the existence of definite changes in the pericardial sac. For obvious reasons there has been, in addition to the gas, a certain amount of exudation, either purulent or hæmorrhagic.

On reflecting the osseous framework of the thorax, the pericardium has usually been seen to bulge forwards in consequence of its distension. This, however, has not invariably been the case, because, as in Begbie's patient, for instance, a free communication may exist, and distension be altogether absent. In cases where the pericardium was inflated and stretched by its contents, the gas escaped on incision with a hissing sound, and had a somewhat foetid odour. So far as can be ascertained by investigation of previous observations, no analysis of the gaseous contents of the pericardium has ever been attempted. When the sac has been freely opened, both the investing and reflecting membranes have presented various degrees of pericarditis. A fibrinous exudation has usually been found upon the epicardium as well as upon the pericardium, and a certain amount of organisation has even been seen forming recent adhesions. A smaller or larger quantity of yellowish, brownish, or reddish foetid fluid has always been present. Some retraction of the lungs and depression of the diaphragm have usually been observed.

SYMPTOMS.—In cases which have permitted the careful observation of the patient from the onset of the disease, rigors have been present, followed by the development of a high but fluctuating temperature, the remissions of which have been, as a rule, attended by profuse perspirations.

Sleeplessness is an extremely common feature of the affection, and evening delirium is also frequently observed.

Breathlessness is always present, and frequently constitutes a marked symptom of the affection. It is, as may be well imagined, frequently accompanied by cyanosis and oedema. Severe pain over the front wall of the chest has been complained of, sometimes amounting to agonising anginous attacks. Palpitation and syncope have also been frequently observed.

The pulse is empty, compressible, and irregular. On inspection of the chest a slight bulging of the præcordia has been noticed, but this seems to be a somewhat rare appearance, and the most striking feature has in most cases been the disappearance of the visible pulsation of the heart. On palpation, feebleness or absence of the cardiac impulse is the prominent symptom. Percussion gives most striking phenomena. All observers are at one in regard to the tympanitic character of the percussion sound, and it has been possible for some of them—Stokes, for example—to determine not only this tympanitic character, but to find sometimes a distinct cracked-pot sound. Perhaps the most important point is, that on changing the position of the patient the character of the percussion sound varies. While the patient is in the recumbent position, the tympanitic percussion sound may be heard over almost the entire præcordia, but in a sitting posture the lower portion becomes dull on account of the change in the position of the fluid and gas. The extent of the area over which the tympanitic sound may be heard has in some cases been co-extensive with the præcordia itself. In other cases it has only been over its lower portion, *i.e.* over the lower portion of the sternum and adjacent cartilages, while the upper præcordial area has given as usual a percussion sound of absolute dulness. On auscultation the sounds observed are of the most characteristic nature. Splashing noises like those of a water-wheel, or of a churn, occurring periodically along with the pulsations of the heart, and therefore rhythmic or arrhythmic according as the action of the heart is regular or irregular, form a most striking combination of sounds. These sounds have a distinct metallic character, and appear to echo within a closed space. Sometimes other sounds are present also. There may, for instance, be a certain amount of friction, and there may also be endocardial murmurs. These sounds,



however, are entirely overshadowed by the wonderful splashing noises. The sounds produced by the pulsation of the heart have occasionally been heard at a considerable distance from the chest. Such was the case in the patient described by Stokes, and Laennec laid considerable stress upon this fact. It might be expected that in cases of pneumopericardium, succussion sounds might be produced, but in the whole literature connected with the subject no observation of this kind has been recorded. The physical signs in pneumopericardium have frequently been seen in patients where the pericardium has been opened for the treatment of pericardial affections.

In most of the undoubted instances of pneumopericardium death has rapidly supervened. A few hours have in some cases been sufficient to produce a fatal result, but two or three days have been more commonly observed to intervene. When death has occurred it has usually been the result of cardiac failure, probably produced by a paralytic myocarditis.

So far as is known to me from a careful survey of the literature of the subject, no case of fistulous pneumopericardium has recovered. On the other hand, traumatic pneumopericardium has sometimes been followed by recovery, and in the cases presenting this favourable termination, the gas has been observed to disappear with a rapidity almost marvellous. Many of the instances of pneumopericardium described as occurring spontaneously are invested by such an atmosphere of what may charitably be termed romance as to merit little criticism.

Brown recorded an interesting case in which a patient, consequent upon a heavy fall of earth upon him, manifested frequent, painful respiration with coughing and hæmoptysis, accompanied by intense pain in the cardiac region. On examination there was a loud double friction sound accompanied by a bubbling and splashing noise which could be heard two inches from the chest wall. The diagnosis was a rupture between the pericardium and the lung. There was little pyrexia, and the pulse and respiration gradually improved day by day, the splashing and bubbling disappeared by the fourth day, the rough friction sound persisted for three weeks, but after the expiration of that period the patient was perfectly well.

DIAGNOSIS.—The recognition of pneumopericardium cannot be regarded as presenting any considerable difficulties. The tympanitic percussion sound over the upper part of the præcordial region, or over the entire area, according to the posture of the patient, and the striking combination of splashing and metallic sounds on auscultation, are absolutely diagnostic. It is sometimes held that a pneumothorax on the left side, or a large pulmonary cavity close to the heart, might be mistaken for pneumopericardium, but this is in the highest degree unlikely. The auscultation of the breath and voice sounds as well as the presence of an approximately normal size of percussion dulness—abnormal probably, however, in its position—are sufficient to distinguish the conditions. The chief difficulty lies in the possibility of mistaking a dilated stomach for pneumopericardium. In some cases of gastric dilatation an area of tympanicity may be determined in the lower portion of the præcordia, more especially on forcible percussion, and on auscultation the heart movements are sometimes accompanied by splashing sounds. It is to be noted that the upper part of the præcordia in such cases is never tympanitic, and that on causing the patient to assume a sitting posture, the splashing sounds disappear.

PROGNOSIS.—Pneumopericardium is one of the most serious affections of the circulation, but the prognosis is much graver in fistulous cases than in those which take their origin in traumatism. In the former, the conditions under which the lesion takes place are in themselves of a nature calculated to lead to a fatal termination, such as a septic abscess opening into the pericardial sac, or a cancer of the œsophagus causing erosion into the sac. In the latter class of cases the wound may heal up rapidly, and the air be speedily absorbed. If the wound, however, occurs in connection with any tissue which has been affected by a diseased process, as in the case just described, the consequences are apt to be more serious.

TREATMENT.—In pneumopericardium caused by a communication with some adjacent focus of disease, the treatment must be simply palliative; but in traumatic cases, strict anti-septic treatment must be adopted at the earliest period possible, while the general condition of the circulation is

watched with a view to obviate any tendencies to cardiac failure. In any cases where pericarditis constitutes a prominent feature, it must be treated on the general principles already laid down, and if there be much exudation, particularly of a purulent character, its removal by appropriate means must be employed.

CASE 11. *Traumatic Pneumopericardium*.—Once, and once only, has any case of pneumopericardium come under my own personal observation. The occurrence took place during my student days. A patient was admitted to the old Royal Infirmary suffering from pleurisy with effusion. As the effusion did not yield to ordinary medical treatment, aspiration was resolved upon, and, in the absence of the resident physician, it was carried out by his substitute. On the conclusion of the aspiration it was found that the condition of the patient had, instead of improving, become worse. The dyspnœa, which had been expected to diminish after the removal of the pleural effusion, became exaggerated. The patient was unable to lie down except for short periods and at long intervals, and the pulse became empty, compressible, and irregular. On examination of the heart the explanation of the change in the condition of the patient was obvious. One of my friends, at that time attached to the ward in which the patient was, requested me, at that time a clinical clerk in an adjacent ward, to see the case, and its clinical features have been stamped upon my memory ever since.

The patient was seated in bed, leaning forward so as to rest upon his knees. His countenance was of a livid pallor, with a deep bluish tinge of the lips, nostrils, and ears. Drops of perspiration stood upon his forehead, and he panted violently for breath. The radial pulse showed an emptiness of the arterial system and a low pressure of the blood. It was extremely frequent and very irregular. There were no characteristic appearances on inspecting the thorax, all local phenomena connected with the præcordia being overshadowed by the severe dyspnœa, and on palpation the only characteristic feature was the extreme indistinctness of the cardiac impulse. On percussion, almost the entire præcordial region gave a ringing tympanitic percussion sound, but at the lower part there was absolute dulness. Auscultation revealed sounds which were very striking. The heart sounds themselves were scarcely audible, but with every movement of the heart there was a loud splashing, extremely like that produced by a churn, and this was followed almost every time it occurred by a distinct metallic tinkling, closely resembling that of fluid dropping in a closed space, such as is to be made out occasionally in large pulmonary vomicæ and in pneumothorax.

It was only too clear that in this case, by some regrettable misadventure, the pericardium had been punctured by the aspiration needle. The course of the affection in this case

was extremely rapid, and the patient died on the second day after the unfortunate occurrence. The dramatic circumstances attendant upon the evolution of the symptoms in this case place it in the same category as that to which belong the instance described by Walshe, in which the juggler perforated his pericardium with a pointed instrument, and that described by Aran, in which the pericardium was punctured by means of a trocar.

### PERICARDIAL TUBERCULOSIS.

Corvisart recognised the existence of tubercle in affections of the pericardium, and Laennec and his successors extended his observations. In recent times a large number of authors have devoted attention to the subject. Simple pericarditis, that is to say, pericarditis in tubercular subjects which is unattended by the formation of the characteristic lesions of tubercle, is not at all an infrequent affection, and it is not to be confounded with tubercular pericarditis and pericardial tuberculosis. In these two latter conditions there are characteristic tubercular lesions of the pericardium. Tubercle of the pericardium may in rare instances be primary and affect the pericardium alone, as in a very interesting observation recorded by Virchow. It is, however, very much more frequent as a secondary process either taking origin from extension of neighbouring organs, or as part of a general infection.

ETIOLOGY.—Tuberculosis of the pericardium may affect every age, but it is more common between the ages of 15 and 30. Duckworth has recorded a case occurring in a child of eight months, while Lee Dickinson and Rolleston have met with instances in children aged nine months. Lejard, on the other hand, has described a primary case in an old woman of 88. According to Osler, it is considerably more common in men than in women.

Primary tubercular pericarditis can only be explained on the assumption that, as in the cases occurring in very young children just mentioned, the patients have been born with the disease present, or that, as is likely in older patients, there has been an inherent weakness or some preceding affection ;



the pericardium may have thus been prepared for the growth of the tubercular bacillus, which has in some obscure way obtained entrance to the sac.

Secondary tubercular pericarditis is most commonly associated with tubercular affections of the pleura or lung, or of the mediastinal or bronchial glands. The latter glands more especially seem to be a source of infection, and many cases which have been regarded as primary, are in reality due to a secondary implication, having origin in an unnoticed tuberculosis of these glands. The course followed by the infective process in some instances is rather obscure, as, for instance, in a case recorded by Schrötter, where tuberculosis affecting the epicardium and the myocardium was associated with no other lesion save a single tubercular mass in the apex of the right lung.

Tubercular pericarditis as part of an acute general tuberculosis only shows itself, according to Osler, when the other serous sacs are also implicated, and it may be termed, with Strümpell, a tuberculosis of the serous membranes.

MORBID ANATOMY.—The appearances presented by the pericardium after death fall into two groups. There is, in the first place, the form which may be strictly termed tubercular pericarditis. The appearances in this group show a certain amount of exudation of fibrin, and of fluid. The fibrinous exudate goes on to a varying degree of organisation, and in the fibrous tissue so arising is the development of tubercular nodules. Within the organising tissue giant cells may be seen on microscopic examination, as is well shown in the illustration, Fig. 126. In some cases of tubercular pericarditis there are no tubercles. One case of much interest is narrated by Kast, in which pericarditis had its origin in a tubercular affection of an adjacent mediastinal gland, and the purulent exudation within the pericardial sac contained tubercular bacilli, but no tubercular lesions were present. There is also, in addition, the much less common form of affection which may be termed pericardial tuberculosis. This in its turn presents two perfectly distinct forms of affection—miliary tubercles may be developed along the small blood vessels, ramifying in the epicardial tissues, and therefore seen most

distinctly in the sulci ; or larger tubercular masses, of grayish tint externally, and with a yellow caseating centre, may be found in some part of the membrane, more particularly about the base of the heart.

In all cases where tubercle is present there is a great



FIG. 126.—Chronic tubercular pericarditis ( $\times 50$ ). *a*, Small tubercular nodules in the epicardium ; *b*, giant cells ; *c*, organising tissue ; *d*, fibrin ; *e*, muscle fibres.

tendency to hæmorrhage. Sometimes superficial ulceration of the pericardial or epicardial structures is seen, and this process may be so serious as to involve, as in an instance recorded by Eichhorst, perforation of the cardiac muscle and rupture of the heart.

**SYMPTOMS.**—Tubercular affections of the pericardium may be absolutely latent throughout their whole course, so that,

unsuspected during life, they are discovered on post-mortem examination.

There are most commonly general symptoms of tuberculosis, excessive fluctuations of the temperature, profuse perspirations, emaciation, and weakness, in short, the development of hectic. The direct evidences of the affection must be sought for, as they are very rarely present in such a form as to attract attention. When present, the local symptoms and physical signs closely resemble those found in an ordinary case of pericarditis. The distinguishing feature of the local manifestations of the disease is the tendency towards chronicity, so that there is much more persistence of the clinical features.

DIAGNOSIS.—The recognition of the tubercular nature of a pericardial affection may be comparatively simple from the combination of unmistakable physical signs of pericarditis with the general features attendant upon tuberculosis. The diagnosis will be rendered easier if tubercle is present in some of the adjacent organs. In doubtful cases in which symptoms of chronic pericarditis remain for a considerable period, it may be advisable to have recourse to aspiration in order to discover the nature of the exudation.

PROGNOSIS.—The prognosis in tubercular affections of the pericardium is always unfavourable, as the disease almost invariably leads to a fatal result, either by cachexia or by some less common termination, such as rupture of the heart. There is no doubt a considerable proportion of cases in which, on account of fibrous changes, a certain amount of recovery ensues, but in such cases the tubercular process follows its course in other organs, and the inevitable result is not far off.

TREATMENT.—The treatment of tubercular affections of the pericardium should be carried out on general principles. An attempt to antagonise the tubercular tendency is necessary, while every means is to be adopted to support the strength of the patient. In particular, such methods as avail to avert cardiac failure should be resorted to. If there should happen to be such an amount of exudation as to interfere with the functions of the heart, it may be necessary to have recourse to aspiration or even to free incision.

CASE 12. *Tubercular Pericarditis*.—L. D., aged 19, engineer, was under my care in the Royal Infirmary, on account of cough and pain in the chest. No family tendencies to disease were present, and the patient's general surroundings had been satisfactory. His previous health, save for the ordinary children's diseases, had been good until eighteen months before admission, when, in consequence of a fall, the left knee was injured, and he was lame for about a month. The knee had always since then been stiff, and ankylosis was threatening. During the month of April the patient suffered from what he was told was inflammation of the right lung, followed by a pleuritic attack, which in turn was succeeded by bronchitis. During the month of July he was sent to Musselburgh in order to seek the benefits of change of air, but as he did not improve he came into the Infirmary. The patient on admission was seen to be a pale, slender youth. His evening temperature rose to about  $102^{\circ}$ , while it was very often subnormal in the morning. The patient's tongue was loaded with a creamy fur, his teeth were bad. He had suffered from diarrhœa before admission, and this continued after he entered the ward. The abdominal viscera presented no abnormality.

The pulse varied considerably in frequency, but it was usually about 100 per minute. The vessel was empty, the blood pressure was low, and well-marked diastolic pulsation was shown. Inspection showed pulsation in the third and fourth intercostal spaces. On palpation the apex beat was felt in the fifth intercostal space three inches from the mid-sternal line. On percussion the right border of the heart was found to be two inches, and the left border of the heart three and a half inches, from mid-sternum. On auscultation, the heart sounds although feeble were heard to be perfectly clear, but they were accompanied by pericardial friction, which extended over the entire præcordia, but was most intense in the third and fourth left intercostal spaces. Over the whole of this area it was characterised by a threefold rhythm, and at the end of expiration it became much more intense. There was a muco-purulent sputum with a tendency towards nummular characters, and on microscopic examination it was found to contain numerous tubercular bacilli. There was flattening of the chest in both infra-clavicular regions, over which, as was also the case in the supra-spinous areas posteriorly, the vocal fremitus was greatly increased. These changes were more pronounced on the left side. On percussion there was dulness in these regions, more particularly on the left side. On auscultation of the left infra-clavicular and supra-spinous regions the character of the breath sounds was bronchial. The expiration was prolonged, and pectoriloquy was present. There were occasional crepitant râles, and sometimes sibilant rhonchi. On the right side in the same regions the breath was of puerile type with prolonged expiration. The vocal resonance was exaggerated, and there were crepitations and sibilations. One most important point in the case was that in the second and third left intercostal spaces there was distinct pleuritic friction. There were no symptoms calling for remark connected with any other system of the body.



The treatment adopted was the administration of cod-liver oil and iodide of iron, together with the external application of iodine. Perfect rest and ample food were enjoined. In two or three weeks the patient was discharged with no evidences of the pericardial affection, which had become latent.

This case furnished a good example of tubercular implication of the pericardium consequent upon a tubercular disease of the lung and pleura.

### PERICARDIAL SYPHILIS.

Syphilitic implication of the pericardium is even less common than in the case of the myocardium, and only a few exceptional instances are on record. Lancereaux observed gumma of the pericardium, without other cardiac lesions, associated with a large syphilitic mass in one of the lungs. Wanitschke, in a newly-born child, affected by hereditary syphilis, saw a characteristic affection of the pericardium associated with a large mass in the upper lobe of the left lung, which, through adhesions with the parietal pericardium, led to a sero-fibrinous pericarditis. The whole subject has been recently studied by Mraček, whose observations and investigations present the best picture of the affection as it affects the pericardium.

**ETIOLOGY.**—In most instances the implication of the pericardium takes place as a late secondary or tertiary manifestation of the disease, but, as has just been seen, it may have its origin in congenital syphilis.

**MORBID ANATOMY.**—The formation of characteristic gummata is a rare feature of the disease; more frequently there is infiltration leading to the formation of fibrous tissue, so that there are adhesions between the pericardium and epicardium. These very rarely lead to any considerable obliteration of the sac, and they are not infrequently attended by a certain amount of fluid exudation.

**SYMPTOMS.**—Such affections have for the most part remained unnoticed during life, and have only been found on post-mortem examination. The affection therefore presents to some extent a contrast to syphilitic lesions of the endocardium

and myocardium, which sometimes lead to characteristic symptoms.

DIAGNOSIS.—The determination of a syphilitic lesion of the pericardium can only be possible when, in addition to proof of the existence of syphilis, there are also physical signs of pericarditis.

PROGNOSIS.—This must entirely depend upon the amenability of the disease to treatment, and it will be the more favourable the sooner symptoms of improvement set in under appropriate remedies.

TREATMENT.—Since the pericardial implication occurs in the late secondary and tertiary stages, iodide of potassium is the most useful remedy, but it is sometimes rendered more efficacious by the addition of perchloride of mercury, while one of the simpler mercurial ointments is applied externally to the præcordia.

### PERICARDIAL NEOPLASMS.

Cancer and sarcoma of the pericardium are by no means common; judging by my own experience, they are less frequent than similar neoplasms of the endocardium.

ETIOLOGY.—Primary cancer and sarcoma of the pericardium are extremely rare indeed; in fact, the only instance which appears to have been absolutely established is one observed by Förster. Secondary manifestations of these affections are somewhat less infrequent, and they have usually been found in connection with malignant disease of the mediastinal glands, pleura, lung, or œsophagus. Occasionally they have followed upon the development of the disease in distant organs.

MORBID ANATOMY.—New formations affecting the pericardium present the characteristic structural features of the disease which may be present, accompanied by a certain amount of infiltration of the serous membrane and of the subserous textures. There is in every case some fluid in the sac, which may be purely serous, but is much more commonly hæmorrhagic, and may even be purulent. In cases where the new formation has caused compression of some of the cardiac veins, the amount of transudation may be considerable.

Among the uncommon affections of the pericardium is actinomycosis. Cases of this kind have been described by Münch. In these cases the parasitic invasion has been associated with lesions in other parts, and the possibility that the pericarditic complications were of this nature was in this way shown.

As pathological curiosities, hydatid cysts of the pericardium have been recorded by Habershon, Landouzy, and Bernheim. None of these cases were recognised during life; they were only discovered on post-mortem examination. If any pericardial symptoms were present in a case of hydatids in other organs, the recognition of the nature of the disease would be rendered possible.

Bouchard, about thirty years ago, described the presence in the pericardial sac of fringes somewhat like those found in the synovial membranes. These sometimes become detached and form free bodies in the sac.

**SYMPTOMS.**—New formations of the pericardium are usually to be regarded as pathological curiosities, inasmuch as the effects produced are inconspicuous, so that their detection is impossible during life. There may, however, be symptoms of pericarditis attended even by characteristic physical signs, and accompanied by the general appearances of a malignant cachexia.

**DIAGNOSIS.**—The probability that any pericardial affection had its origin in some new formation, would be strengthened by the existence of the presence of such a disease in some other part of the body, and by interference with the functions of such adjacent organs as the trachea or œsophagus.

**PROGNOSIS.**—The prognosis in all cases of this kind is absolutely unfavourable.

**TREATMENT.**—It is impossible in cases of malignant disease of the pericardium to do more than meet the symptoms and obviate the consequences of the disease.

## HYDROPERICARDIUM.

The pericardial sac is found in almost every instance after death to contain a varying but moderate amount of clear

yellowish fluid, which is commonly termed the liquor pericardii. The amount of this fluid after death has probably but little relation to that which is contained by the pericardium during life, and it appears to be a well-established fact that when the death agony has been prolonged, the amount of fluid is increased. The quantity usually found is on an average from 5 to 10 c.cm., but it may rise to 90 or 100 c.cm. When this amount is surpassed it may be regarded as the result of pathological conditions.

Hydropericardium or pericardial dropsy consists in a serous transudation which accumulates in the sac, and is analogous to hydrothorax and ascites. By the earlier authors of modern times the presence of fluid in the pericardium was very commonly recognised, as for instance by Lower, Schellhammer, Senac, and Morgagni, but as in those earlier days no distinction was made between exudation and transudation, the observations are now of comparatively little value.

ETIOLOGY.—Hydropericardium is necessarily always a secondary affection, and has its origin in the various conditions producing œdema which were previously considered. It is produced by general venous stasis, having its origin in valvular disease or myocardial changes, and it may arise in a more remote manner from affections of the lungs, pleura, or kidney. Local venous stasis may be produced by pressure upon the cardiac veins in consequence of some new formation connected with the heart, pericardium, or mediastinum. Pericardial dropsy may also be produced by various cachectic conditions. Certain authors, and more particularly von Bamberger, have attempted to prove the existence of what has been called "hydropericardium ex vacuo." The theory is that in consequence of shrinking of the lung or atrophy of the heart, the tendency to produce a vacuum has led to the development of transudation. As has already been sufficiently pointed out, there is under normal circumstances always a negative pressure in the pericardial sac, and yet the amount of fluid which it contains is small. Such a supposed cause of transudation cannot therefore be accepted, and Friedreich was perfectly right in declining to entertain its possibility. A transudation can only occur in consequence of such disturbances of the serous



membrane, the blood vessels, or the blood itself, as have in a previous section been fully described.

**MORBID ANATOMY.**—The fluid found in the sac in hydropericardium is clear, and of a yellowish or greenish tint, with a dichroic tendency. Sometimes it has a reddish colour from the presence of blood, or, if the broken-down colouring matter of the blood is present, it may be of a dirty brownish hue. The specific gravity of the fluid is usually about 1015. Sometimes the fluid is somewhat flocculent, and, when this is the case, endothelial cells are found on microscopic examination of the deposit which is thrown down on standing. These epithelial cells are usually granular. A small amount of round cells, also usually granular, may be seen, and there are not infrequently small fibrinous masses. According to von Dusch and Eichhorst, cholesterin crystals are not infrequently present. The quantity of the transudate not uncommonly reaches 1000 c.c., and in a case recorded by Corvisart there were actually, unless some error has crept into the record, 4000 c.c.

On examining the pericardium after death, it is found to be distended according to the amount of fluid which is present, and to show distinct fluctuation. The serous surfaces are smooth, shining, and free from any fibrinous exudate. They are usually pale on account of the pressure on the vessels of the membrane. The pericardium is sometimes very thin, but at other times—and this is more particularly the case when the accumulation has been gradually developed—it may be considerably thickened. The sub-epicardial fat to a considerable degree disappears, and the subserous tissues are pale and macerated, or even œdematous. This œdematous condition may also be found in the adventitia of the coronary vessels, and of the great vessels, at the base of the heart. The right ventricle and right auricle are often considerably dilated, and the muscular tissue of the heart in general is pale and flabby. There is frequently œdema of the cellular tissues of the mediastinum, and compression of the lower lobe of the left lung is very commonly present.

**SYMPTOMS.**—Since hydropericardium is in most instances only part of a great complex of pathological conditions, the

symptoms which it produces are usually overshadowed by the clinical features belonging to the primary affection with which it is associated. It may be said to step in as the closing link of a pathological chain, and once it has made its appearance it unites with the other morbid conditions to form a vicious circle of fatal import.

Hydropericardium is not necessarily associated with any pyrexia; it is not, indeed, uncommon to find it attended by subnormal temperature. It at times gives rise to sensations of oppression in the chest; dyspnœa, cyanosis, and œdema are also of almost constant occurrence in at least some degree. Attacks of syncope are often found. Patients in whom dropsy of the pericardium has occurred often present various nervous symptoms. A soporose tendency may be constantly present, or there may be, more especially in the evening, mild muttering delirium. The attitude assumed is very often an upright one, or the patient leans forward, resting his arms upon the knees. On examination, the pulse is usually empty, compressible, frequent, and irregular. The veins of the neck are turgid. There may be a fulness of the præcordia and a widening of the intercostal spaces. The cardiac impulse is imperceptible even when the patient leans forward. The vocal fremitus over the præcordia is diminished. There is an increased area of cardiac dulness, which assumes the form which has been seen to be characteristic of a pericardial exudation. The heart sounds become extremely feeble, and if any adventitious murmurs have previously been present, their intensity is also diminished. There never is, under any circumstances, a friction sound. On examination of the abdomen, the liver is often found to be enlarged and ascites to be present. The amount of urine is diminished, and it often contains albumin. The bases of the lungs may, by means of muffling of the percussion sound and crepitations, reveal the presence of œdema pulmonum, or by dulness of the percussion sound, and absence of voice and breath sounds, that of hydrothorax.

DIAGNOSIS.—The diagnosis of hydropericardium must rest upon the presence of signs of effusion in the absence of any proof of pericarditis. It is usually facilitated, however, by the existence of sufficient causes to produce the effusion.

PROGNOSIS.—The prognosis in hydrothorax is entirely dependent upon its etiology. Some of its causes are to some degree remediable, as in the case of acute nephritis, and when this is the case the outlook is more hopeful, but in the overwhelming proportion of cases the etiology of the affection renders it as grave as possible.

TREATMENT.—Since hydrothorax is practically a symptom of some primary affection, its management depends upon the treatment of the original disease; but in addition to the appropriate means for such a condition, the administration of dry food, and the employment of diuretics and purgatives, are strongly indicated. If the hydrothorax so aggravates the primary condition as to produce urgent symptoms, it may be necessary to have recourse to paracentesis.

### HÆMOPERICARDIUM.

Those forms of pericarditis in which blood is present have already been referred to, more particularly the forms which occur in the course of scurvy, cancer, and tubercle, as well as in renal diseases and alcoholic conditions. Hæmopericardium is in no way connected with such affections, and in its structural alterations and clinical features presents no relationship to hæmorrhagic pericarditis.

ETIOLOGY.—The causes of hæmopericardium may be traumatic, from direct or indirect violence, or pathological, in various diseased processes. Amongst traumatic causes are wounds inflicted by projectiles or sharp instruments, or arising in consequence of a blow or fall; in these two latter cases, however, it is probable that some structural alteration has been present, predisposing to rupture of the heart. Of the pathological causes of hæmopericardium may be mentioned:—rupture of the heart, taking place, as a rule, during some violent exertion, but resulting usually from structural disease; rupture of one of the coronary arteries in profound arterial degeneration; ulceration of the heart or of one of its vessels; rupture of a degenerated aorta or of an aortic aneurysm opening into the pericardial sac—all these are not uncommon examples of the causation of hæmopericardium.

MORBID ANATOMY.—On examination of the pericardium after death, the amount of blood poured out is found to vary very considerably. In cases characterised by rapid development and sudden death, the amount of blood is usually small; instances of slower evolution and more lingering death furnish examples of amounts which may be very considerable. The blood may be entirely coagulated or it may be altogether fluid. The most common condition, however, is one in which a certain amount of coagulation has taken place, but in which some of the blood remains fluid. The examination, it need hardly be added, reveals, in such cases as merit the name of hæmopericardium, the source of the blood. The condition is, as a rule, entirely diverse from those forms of pericarditis in which a certain amount of blood is present, and in which its source is occasionally quite obscure.

SYMPTOMS.—The clinical features presented by patients suffering from hæmopericardium furnish extremely variable pictures. At times the events unfold themselves with a swiftness that is appalling—the patient, one moment, to all appearance in ordinary conditions of health, and, it may be, engaged in some occupation; the next moment, lying collapsed and pulseless. On the other hand, the opposite extreme shows a somewhat protracted and lingering passage into asystole. Sometimes at the onset of the affection the patient complains of a sudden sharp pain in the præcordia, or of a feeling as if something had given way. Giddiness and faintness are apt to follow upon such sensations, or they may themselves be the earliest manifestations of the disaster which has occurred. The feeling of faintness commonly passes into unconsciousness, while the giddiness occasionally leads to convulsive seizures. The patient usually presents a deathly pallor, sometimes with a cold perspiration bedewing the features. The pulse is empty, feeble, and irregular. On examining the chest there is, as a rule, a total absence of all characteristic phenomena, and only in those forms in which the affection has come on slowly, is it possible to find any physical signs that might be useful. In such cases the area of dulness may be somewhat enlarged.

The most characteristic instances of hæmopericardium which have come under my notice have occurred in consequence



of wounds, ruptured aorta, and ruptured aneurysm. They will be referred to in subsequent sections.

DIAGNOSIS.—The recognition of the nature of the event in hæmopericardium is only possible when the existence of some affection likely to produce it has been previously established, and it can only be determined with any degree of probability when sudden collapse is associated with the physical signs of enlargement of the area of præcordial dulness.

PROGNOSIS.—With few exceptions, the prognosis in cases of hæmopericardium is absolutely hopeless.

TREATMENT.—Therapeutic measures in almost every case are impotent.

## CHAPTER VIII.

### DISEASES OF THE ENDOCARDIUM.

AFFECTIONS of the endocardium have been recognised since the dawn of modern pathology. Senac, Morgagni, and Boerhaave described with greater or less exactness various changes in the lining membrane of the heart, and paved the way for later observers. The association of endocardial lesions with acute rheumatism was suspected by Baillie, and was mentioned by Kreisig; these observers, however, did not lay any particular stress upon the co-existence of the diseases, and it was left to Bouillaud to show the distinct connection between the general disease and the local complication. To Bouillaud is also due the credit of having directed attention to the frequent association of other acute diseases and valvular lesions, while he also introduced the term endocarditis.

The next advance was made by Kirkes, who laid the foundation for our knowledge of the more serious types of endocarditis, and of the embolic origin of septicæmia and pyæmia. His researches were pushed further by Virchow and many subsequent writers. The connection between micro-organisms and endocarditis has been gradually established during recent years. Winge appears to have been the first observer who really made out the presence of organisms in endocarditis, and he was followed by Heiberg and Köster. Klebs stated that many different organisms might be the cause of endocarditis. Weichselbaum and Wyssokowitsch determined the presence of pyogenic organisms in the endocardium, and their investigations were completely verified by the researches of Fränkel and Sängér. The presence of the tubercular bacillus

in endocarditis was mentioned by Cornil and Babes, and this observation has been amply verified and greatly enlarged by Tripier. The pneumococcus was observed by Netter in endocarditis associated with pneumonia, and Weichselbaum has also described this organism in endocarditic affections. The experimental production of endocarditis in animals will be referred to in considering the etiology of the disease, and it need only be said in this place that the observations of Rosenbach and Wyssokowitsch have thrown considerable light upon the origin of the disease.

### ENDOCARDITIS.

Many attempts have been made to classify endocarditis. None of these, however, are perfectly satisfactory. By the classification which is most in vogue, the disease is subdivided into an acute, including malignant and benign varieties, and a chronic form. Such an arrangement on examination breaks down in many ways. All acute forms of endocarditis—and they are many—if not due to, are attended by, the presence of micro-organisms; the same micro-organisms being present in those forms commonly regarded as malignant or infective, and in those which are not. The lesions which are found after death are frequently identical in cases which would be considered as belonging clinically to different classes. The diagnosis of cases which are to be regarded as infective can only be determined by the employment of bacteriological tests after death.

From the clinical point of view the separation of malignant and simple acute endocarditis is a matter of extreme difficulty. Many instances, which appear for a time to be free from any malignant tendency, suddenly undergo a change in their symptoms, and end disastrously, while others, which have set in with the most serious pyæmic symptoms, undergo a favourable alteration in their clinical features, and end in resolution.

In the following pages, therefore, a different classification is adopted. No attempt is made to differentiate the malignant, septic, or ulcerative forms of endocarditis, as regards pathological characters, from the benign, simple, or verrucose varieties, and

the disease is considered chiefly from the clinical side. From this point of view the affection naturally falls into the three groups of acute, subacute, and chronic endocarditis which were long ago adopted in the excellent work of von Dusch.

*Frequency.*—The numerical results given in regard to endocarditis cannot be accepted with the same confidence as is the case with pericarditis, since the diagnostic factors relied upon are far from uniform. The statistics of the Royal Infirmary for the last five years, as given by Gillespie, are of interest if of less value than the corresponding facts relating to pericarditis. In a total of 2368 cardiac cases, 50 were diagnosed as endocarditis—26 males and 24 females, of whom 14 and 18 respectively died, *i.e.* a death-rate of 53·9 and 75·0 per cent. The details are as follows:—



ADMISSIONS.						
Age.	Males.	Per cent.	Females.	Per cent.	Total.	Per cent.
1-9	1	3·8	2	8·3	3	6·0
10-19	5	19·2	7	29·0	12	24·0
20-29	7	26·9	6	25·0	13	26·0
30-39	3	11·5	5	20·8	8	16·0
40-49	6	23·0	3	12·5	9	18·0
50-69	3	11·5	1	4·1	4	8·0
+ 69	1	3·8	...	...	1	2·0
Total .	26	52·0	24	48·0	50	...

MORTALITY.						
1-9	...	...	2	100·0	2	66·6
10-19	3	60·0	4	57·1	7	58·3
20-29	1	14·2	3	50·0	4	30·7
30-39	2	66·6	5	100·0	7	87·5
40-49	4	66·6	3	100·0	7	77·7
50-69	3	100·0	1	100·0	4	100·0
+ 69	1	100·0	...	...	1	100·0
Total .	14	53·9	18	75·0	32	64·0

ETIOLOGY.—Among the *predisposing causes* of endocarditis individual circumstances or personal conditions are of some importance.

Acute endocarditis is essentially a disease of the active period of adult life, its occurrence being most frequent between the ages of twenty and forty. Many statistics have been published with regard to the incidence of acute endocarditis, of which those based upon the results of morbid anatomy are alone of any value. For reasons which will be given in dealing with the symptoms of the disease, statistics founded upon

clinical observation in acute endocarditis are notoriously unreliable.

It has been regarded as a well-established fact that the more serious types of endocarditis are most frequently found amongst women, while the simpler forms are more common amongst men. These facts have been held as true also for children. When acute endocarditis affects youthful patients, its graver forms have been said to be found more amongst girls than boys, and the less serious forms to be more common amongst boys. The recent observations, however, of Kelynack, as well as of Kanthack and Tickell, throw some doubt upon the accuracy of these views. Kelynack found amongst his cases of fatal infective endocarditis that 68 per cent. were of the male, and 32 of the female, sex. Kanthack and Tickell, from the analysis of all the cases of fatal infective endocarditis at St. Bartholomew's Hospital from January 1890 to March 1897, give the following table of 84 cases:—

Age.	Males.		Females.		Total.
Under 10	3	1-5 = 1 5-10 = 2	2	1-5 = 0 5-10 = 2	5
10-20	9	10-15 = 2 15-20 = 7	11	10-15 = 4 15-20 = 7	20
20-30	13	20-25 = 5 25-30 = 8	6	20-25 = 4 25-30 = 2	19
30-40	19	30-35 = 9 35-40 = 10	3	30-35 = 2 35-40 = 1	22
40-50	5	40-45 = 2 45-50 = 3	6	40-45 = 4 45-50 = 2	11
50-60	2	50-55 = 0 55-60 = 2	5	50-55 = 4 55-60 = 1	7
Total	51 =	60·71 per cent.	33 =	39·29 per cent.	

As regards the age, so far as the males are concerned, most of these cases occurred between the ages of 20 and 40 years, namely, 62·7 per cent., while among females most cases occurred between 10 and 30 years, viz. 51·5 per cent., 33·3 per cent. occurring between 15 and 25 years.

Amongst the predisposing causes previous valvular disease is a most potent factor. This was first observed by Paget. Kelynack found it in 80 per cent. of his cases, and Kanthack and Tickell in rather more than 64 per cent. Congenital disease has long been known to act in the same way as acquired valvular affections.

The *exciting causes* of endocarditis fall into the classes of infective processes, and constitutional affections. Direct extension, which has been seen to occur not infrequently as a cause of pericarditis, may practically be excluded as a real factor in the evolution of endocarditis, except in some cases of septic myocarditis.

Many of the acute infectious diseases are connected with attacks of endocarditis. Scarlet fever has been known since the days of Bouillaud as a frequent cause. There are some interesting points regarding the connection between the two diseases. A strong suspicion has long been entertained that very probably the endocarditic sequelæ are determined by a secondary infection through the rheumatic virus. This was more particularly urged by Trousseau and Peter. In such cases streptococci have been detected upon the affected valves, which strongly supports the view of mixed infection. Such observations, however, are of but little import, inasmuch as streptococci are found in almost every variety of endocarditis. Measles is occasionally complicated by the affection, as originally recognised by Bouillaud. The coincidence of the two affections, however, is very rare. Smallpox has been accompanied by endocarditis. To this point Simonet, Trousseau, and Duroziez have paid particular attention, and from their observations it is known that such complications are much more common in the confluent type of smallpox. Influenza, as we know from the recent epidemics, may give rise to, or be accompanied by, endocarditis. The most important work upon this subject is that of Fiessinger. It appears doubtful whether diphtheria can give rise to endo-

carditis. Talamon and Osler, in 138 autopsies of patients dying of diphtheria, never once encountered the condition. The coincidence of the two affections has from time to time been signalised, but probably in such cases double infection has been present. Endocarditis, in the case of patients who have died of diphtheria, has revealed recent changes in the mitral cusps with the presence of various micro-organisms. In erysipelas the affection may arise, and Denucé has described the special organisms of erysipelas in the affected cusps. Endocarditis is rare in enteric fever. The concurrence of the two affections, nevertheless, was notified by Griesinger, and his observations have been corroborated by those of other workers. Girode has been able to determine the presence of the enteric bacillus upon affected valves—a somewhat remarkable fact, seeing that this organism appears to have the greatest difficulty in living in the blood.

Pneumonia frequently gives rise to endocarditis. The clinical fact has been known since the second edition of Bouillaud's work appeared, but experience has varied greatly in regard to the numerical relation. Osler found it in 15 per cent. of his cases of pneumonia. In recent times the characteristic organism of pneumonia has been determined on the cusps by Netter and other observers. It is most interesting to note that endocarditis characterised by the presence of pneumococcus upon the affected cusps has been seen by Weichselbaum and other observers in the absence of any pneumonic affection; the observer mentioned has met with it along with cerebro-spinal meningitis, in which the lesions contained the characteristic cocci, in the absence of pneumonia. In many of those instances the pneumonic coccus is accompanied by other organisms, more particularly by streptococci and staphylococci.

The disease is not by any means a frequent sequel to malarial influences, yet several observers, and more particularly Duroziez and Lancereaux, have insisted on the effects of paludism upon the endocardium. So far the presence of the plasmodium malarie has not been ascertained in the lesions of endocarditis, and the question must remain obscure until such has been done.



Pyæmia in its various forms is fruitful in the production of the affection. In the puerperal condition it was described many years ago by Simpson, and it has in its protean manifestations been recognised by numerous observers as the determining factor in endocarditis.

In gonorrhœa it is seen from time to time, and the connection seems to have been first suspected by Brandès. The gonococcus has been determined on the affected valves, so that the relationship has been placed beyond the possibility of doubt.

Tubercular endocarditis has long been suspected, but only recently found. It is perhaps more common in acute tuberculosis than in any other manifestation of the disease. The lesions have been more particularly studied by Perroud and Tripier.

Endocarditis arises in the course of many constitutional diseases.

Acute rheumatism is the most fertile cause of the affection. This, as already mentioned, attracted the notice of Baillie, and it was more particularly observed by Kreisig; the connection between the two affections, nevertheless, was not thoroughly established until Bouillaud enunciated his famous laws, which, stated shortly, are:—1. That in acute articular rheumatism of a violent type and general distribution, the coincidence of pericarditis and endocarditis is the rule, and the non-coincidence the exception. 2. That in acute articular rheumatism of a mild type and partial distribution, the non-coincidence is the rule, and the coincidence the exception.

Statistics with reference to the frequency of endocardial complications in acute rheumatism have been frequently compiled. In this country the best known statistics are those of Latham and Sibson. The former found that in 136 cases of acute rheumatism the heart was affected in 90 and exempt in 46. Of the 90 cases in which there was a cardiac affection 63 had solely endocardial, and 7 purely pericardial lesions; both membranes were involved in 11 cases; in 9 the case was doubtful. The latter out of 325 cases of acute rheumatism found 130 in which endocarditis occurred, 54 in which both endocarditis and pericarditis were present, and 9 in which pericarditis alone made its appearance; in 3 of which, how-

ever, it was doubtful whether endocarditis was present or not. Continental statistics give extremely variable figures; the statistics furnished by observers such as Bamberger and Jaccoud show a percentage of from 20-30. During childhood rheumatism exercises a more serious influence over the endocardium than is the case in adult life. Every observer has the same experience to relate in this respect. The statistics of West give 61.3 per cent. of heart complications in the course of this disease, while the percentage is 81 according to Cadet de Gassicourt. As was seen by Bouillaud and Trousseau, endocarditis may precede articular manifestations, and this, which has frequently been observed since, can only be regarded as a consequence of the rheumatic poison reversing the usual order of its mode of incidence in consequence of greater endocardial proclivity.

This is not the place to discuss the probable nature of the rheumatic poison, but it may be said that it becomes more and more manifest that it is either of a nature akin to that of those producing the acute infectious diseases, or is attended by such an agent, since its complications are so commonly microbic in origin. The recent researches of Bouchard have gone a long way towards the determination of this important point.

Just as chorea, even in the absence of all overt rheumatic symptoms, may give rise to pericarditis, as shown by Bright, so it may also produce endocarditis. In such cases the toxin products of the rheumatic poison may be held to have a greater affinity for the cardiac and cerebral membranes than for the membranes lining the articulations. In 71 cases of chorea, Roger found that 47 presented diagnostic signs of endocarditis alone, while 19 more had both pericarditis and endocarditis. Similar statistics have been collected by other observers. Chorea is not the only affection in which the rheumatic poison in a marked form occurs. Erythema nodosum was shown by Trousseau to occur with endocarditis, and there cannot be the shadow of a doubt that this skin affection is connected with the morbid agent of rheumatism. Mackenzie's observations on this subject are of great value.

In the gouty diathesis the evolution of endocarditis is

uncommon. It is perfectly true that valvular lesions have their origin in the course of this affection, but such valvular lesions are for the most part of degenerative type, and have little in common with endocarditis. It nevertheless happens that the uric acid diathesis is accompanied by endocarditis, which, however, may be determined by the presence of a microbic poison in no way connected with the gouty affection, save in so far as the latter paves the way for the influence of the organisms. It would, however, be in the highest degree unscientific to deny that faulty chemical processes give rise to products which may set up endocarditis. True, tophaceous deposits upon valves have been seen by Lancereaux and others, but, if connected with endocarditis, they must be of the nature of sequelæ.

Endocarditis is frequently attributed to renal disease, and there can be no doubt of the direct relationship in the case of primary infective nephritis, but when the case of simple nephritis is considered, the matter is by no means so clear. The great majority of endocardial affections in simple nephritis are of the nature of degeneration. There are, nevertheless, many instances in which acute endocarditis takes its origin in the course of such renal disease. In many of these cases the determining cause may be a secondary infection by means of streptococci, for which the renal condition has simply prepared the soil, or it may be that some intercurrent affection, such as acute rheumatism, has taken place; yet, when such considerations have been discussed, it must be admitted with Hanot that there is a possibility of the generation of ptomaines, with the power of producing endocarditis. In renal disease the endocarditis may be acute, subacute, or chronic.

Closely associated with such constitutional and local disorders as have been discussed are the effects of certain chemical products. Alcohol and its congeners belong to this group. The abuse of alcohol has a most hurtful influence over the endocardium, and it at any rate produces a liability to endocarditis in its chronic forms.

In addition to these causes, long-continued muscular strain has an undeniable influence in the production of endocardial disorders. Boerhaave observed that wild deer were very much

more liable to changes in the lining membrane of the heart and great blood vessels than those which were kept in captivity, and he rightly attributed the difference to the much greater muscular exertion undergone by the former. His views were accepted by Morgagni and by Haller, and they may be said to have laid the foundation for our present knowledge in regard to the relations between excessive strain and endocardial or endarterial disease. It is a matter of common observation that certain classes of men who are in the habit of undergoing great physical exertion are liable to such changes—a subject which has during recent times attracted much attention from many workers at heart disease, amongst whom may be mentioned Myers and Clifford Allbutt.

It is necessary before leaving this subject to consider in greater detail some facts relating to bacterial infection.

Micro-organisms, as was shown by Klebs and Köster, are found in all forms of endocardial vegetation. This has been verified by Osler, and more recently placed beyond all possibility of doubt by Fränkel and Sängner. Many different kinds of micro-organisms have been detected. Pyogenic microbes are the most frequent organisms seen in the diseased valves, but many other bacilli and cocci are found, although more rarely. Some special forms have been described, as, for instance, the *bacillus endocarditis capsulatus* of Weichselbaum, the *bacillus endocarditis griseus* of Weichselbaum and Netter, the *bacillus immobilis et fœtidus* of Fränkel and Sängner, and the *micrococcus endocarditis rugatus* of Weichselbaum. Others have been described, but their characters are somewhat less positive than those just mentioned. In most instances only one organism can be detected, but cases have been described in which several different kinds have co-existed. When secondary changes have occurred through embolism, the same organism found in the heart has frequently been found also in the secondary deposits.

A large series of investigations has been carried out in order to discover how these organisms can produce endocarditis. Klebs enunciated the opinion that the organisms circulating in the blood simply acted upon the endocardium by attacking its surface. Köster, on the other hand, was of opinion that



the organisms were carried by the blood into the substance of the endocardium, and produced embolism. This view is beset by difficulties arising out of the facts previously given as to the absence, in great part, of blood vessels in the valves. Wyssokowitsch found that by injecting staphylococci and streptococci into a vein, after wounding the cardiac valves by means of a stylet introduced through a carotid artery, acute endocarditis of the valves was produced, and it was shown previously by Rosenbach, as well as by Wyssokowitsch, that the wounding of the valves by means of a sterilised instrument did not produce endocarditis. Ribbert, however, found that the introduction of staphylococci by a vein of the ear was in itself sufficient to set up endocarditis, and Dreschfeld was successful in cultivating organisms from the vegetations of endocarditis destitute of malignant or ulcerative features; these organisms, when introduced into the rabbit, produced most extensive vegetations on the mitral and aortic valves. The same streptococcus was found in the vegetations and in the heart from which the organisms were derived. Prudden has summarised much of this recent work.

It seems clear that in certain instances the blood may contain a poison whose virulence is sufficient to produce endocarditis without previous damage; in other cases previous lesions have, nevertheless, been in existence. This is not only true experimentally, but also as regards the results of morbid anatomy, for acute endocarditis is very frequently grafted upon old-standing valvular lesions. It seems also probable that the lymph circulating through the interstices of the valve may be unable to supply them with proper nourishment, and in this way they may be predisposed to infection.

A new phase regarding the poisons in acute endocarditis has begun with the observations of Sidney Martin. Chemical examination of the blood and of the spleen in a case of infective endocarditis with staphylococci which could be cultivated, revealed two chemical substances—the one, a proteid, consisting of proto-albumose and deutero-albumose; the other, a non-proteid body with strong acid reaction. The albumoses, when injected into animals, gave rise to pyrexia, varying in degree with the amount employed. The coagula-

bility of the blood was lessened, and, after death from overdoses, fatty degeneration of the heart was found.

The conclusions of Hanot regarding the pathogenesis of endocarditis are worthy of careful consideration. Whether in respect of the special nature of the microbe, of difference in the intensity of its action, or of varying power of resistance, there may be two modes of development. In the one case, the lesions in endocarditis consist of changes with a tendency to localise and concentrate themselves, to resemble more and more the fibrous tissue of a scar, and, in this way, to a certain degree, to lose infectious characters. Such is attenuated infectious endocarditis or benign endocarditis of the physician, which allows the individual to pass from an acute to a chronic condition. On the other hand, the endocardial lesion may preserve in a high degree its original infectious character, when the morbid tissue has a tendency to break down and be resolved into particles, which assist in transporting the infectious agent and penetrating deeply into the organism. Such is malignant endocarditis, infectious and infecting, brought first into prominence by Senhouse Kirkes.

The analysis of eighty-four cases by Kanthack and Tickell has been already quoted as regards predisposing causes. With reference to direct causation their interesting statistics may again be laid under contribution.

In all but ten cases, old cardiac disease, or an infective lesion, accompanied the endocarditis. Of these ten cases there was malignant disease in four, and a possible pneumonia in one, so that only five cases remain which can be considered as uncomplicated, *i.e.* as primary infective endocarditis, not preceded by either cardiac lesions, or infective processes, in other parts of the body. In one of these five the head had not been examined, so that the condition of the middle ear was not ascertained.

If we turn our attention to the complicated cases, we learn that in the eighty-four there was pre-existing cardiac disease in fifty-four (or 64·27 per cent.), namely, thirty-two males (or 62·7 per cent. of all males), and twenty-two females (or 66·6 per cent. of all females). Of these fifty-four cases,

sixteen (or 29·8 per cent.) were accompanied by recognised infective processes (not counting acute rheumatism or chorea as such), while the remaining thirty-eight (or 70·2 per cent.) showed no such lesions. These cases have been arranged in the following table :—

INFECTIVE ENDOCARDITIS PRECEDED BY OLD CARDIAC DISEASE.

Without Infective Processes.		With Infective Processes.											
Male.	Female.	Pneumonia.		Pyogenic.		Empyema.		Vaginal and Uterine.		Bronchiectasis.		Influenza.	
Total, 23=45 per cent. of all males, or 71·87 per cent. of all males with cardiac disease.	Total, 15=45·45 per cent. of all females, or 68·18 per cent. of all females with cardiac disease.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
		3	4	3	1	1	...	...	2	1	...	1	...
Total=38, or 70·2 per cent. of all cardiac cases.		Total=16, or 29·8 per cent. of all cardiac cases. (Males = 28·13 per cent. ; females=31·81 per cent.)											

Bronchiectasis, empyema, vaginal or uterine affections, and also influenza, must be regarded as pyogenic sources of infection. Exception might be taken to the inclusion of influenza in this group since it is caused by a specific bacillus, but pyogenic organisms are always present in the lungs in this disease, and in the case given in the table streptococci were found both in the lungs and in the heart's blood. Hence among the infective processes there were found nine cases of pyococcus infection, and seven cases of pneumococcus infection.

In thirty cases old cardiac changes were absent, that is, in 35·73 per cent., but in twenty of these, infective processes were discovered which had appeared either before or with the infective endocarditis. Of the ten where infective processes were not evident, four were complicated by malignant disease and one possibly by pneumonia; it may therefore be said that twenty-five presented certain or probable sources of infection,

while in five none were found. Among the sources of infection were pneumonia and pyogenic lesions, including otitis media, puerperal affections, and typhoid ulceration. All these cases have been arranged as follows:—

INFECTIVE ENDOCARDITIS WITHOUT PRE-EXISTING CARDIAC DISEASE.

Without Infective Processes.			With Infective Processes.													
M. F.		Remarks.	Pneu- monia.		Pyo- genic.		Em- pyema.		Vaginal and Uterine.		Otitis.		Tuber- cle.		Ty- phoid.	
M.	F.		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
5	5	4 were cases of malignant disease, 1 possibly pneumonia, in 1 head not examined.	M. 5	F. ...	M. 3	F. 3	M. 1	F. ...	M. ...	F. 2	M. 2	F. 1	M. 2	F. ...	M. 1	F. ...
Total = 10, or 33·3 per cent. of all cases without cardiac disease.			Total = 20, or 66·6 per cent. of all cases without cardiac disease. (Males = 14 ; females = 6.)													

Pneumonia occurred five times, and pyogenic infections thirteen times, including vaginal and uterine infections, otitis media, empyema, and tuberculosis. The latter may fairly be included, since it is well known that the tubercle bacillus is generally associated in the lungs with pyococci; only one case, however, can be added to the pyogenic infections, because in one case there was a tubercular endocarditis. It may also be remarked that in the typhoid case both typhoid bacilli and cocci were found in the heart's blood. It is noteworthy that in those cases in which old cardiac disease was absent, evident sources of infection were far more numerous than in those cases in which old valvular lesions were present; for the former group there is a percentage figure of 66·6, and for the latter of 29·8.

Averaging together all the cases with a manifest infective process, whether cardiac disease was present or not at the same time, they amount to thirty-six, or 42·85 per cent. of all the cases collected. These are tabulated as follows:—



## ENDOCARDITIS WITH INFECTIVE LESIONS.

Pneu- monia.	Pyogenic.	Otitis.	Vaginal and Uterine.	Empyema.	Bronchi- ectasis.	Tubercle.	Influenza.	Typhoid.
12	10	3	4	2	1	2	1	1

Adding all the pyogenic lesions together, they amount to twenty-four, or 28·56 per cent. of all cases, while pneumonia occurred in 14·25 per cent. of all cases, otitis in 3·55 per cent.

One of the most interesting points is, that an antecedent pneumonia occurred in 14·28 per cent. of all instances of infective endocarditis. In these cases which appear in the course of croupous pneumonia, the pneumococcus is the cause of the endocarditis, and can readily be separated after death. It seems, further, that a pre-existing cardiac lesion attracts the pneumococcus, for, of twelve cases of pneumonic endocarditis, seven were complicated by old cardiac disease.

Infective endocarditis is by no means common in typhoid fever; on the contrary, it is distinctly rare. When it occurs, either streptococci and other pyococci or typhoid bacilli have been found in the heart's blood. Kanthack and Tickell found the Eberth-Gaffky bacillus in the heart's blood, not in pure culture but along with streptococci. Similarly, in those out-of-the-way cases of endocarditis appearing during the course of diphtheria or gonorrhœa, the Klebs-Löffler bacillus, the Neisser coccus, or streptococci and other pyococci, may be present, and there may be a distinct tubercular endocarditis; mixed infections are common in all these cases.

There may be, therefore, when an endocarditis appears in the course of an infective fever: (1) homologous, or (2) heterologous, or (3) mixed infection of the endocardium.

MORBID ANATOMY.—Acute endocarditis is much more common on the left than on the right side of the heart. No adequate reason for the great disproportion existing between the number of instances affecting the left and right sides of

the heart has yet been adduced, but it is generally believed that there are two factors causing the disparity. The greater pressure on the left side must render the endocardium more liable to changes, and in addition the arterialised condition of the blood on the left side must be more favourable to the development of micro-organisms. Amongst the more serious varieties of acute endocarditis—those often clinically termed malignant—however, a somewhat larger proportion affect the right side, and it seems probable that the reason for this lies in the fact that septic poisons on entering the system are more likely to encounter the right side of the heart in the first instance.

Bramwell is of opinion that endocarditis affects the right



FIG. 127.—Acute endocarditis of the mitral valve. One of the musculi papillares has been cut through.

side of the heart much more commonly than is usually believed, and he further holds that acute changes in the tricuspid valve frequently subside altogether and entirely disappear. As the evidence, however, upon which he relies for the diagnosis of endocarditis cannot be considered absolutely satisfactory, the point must be regarded as far from proven. It must be remembered that Albini's bodies are very common. These consist of fusiform cells, elastic fibres, and connective tissue, and are most frequently found on the borders of the auriculo-ventricular cusps. They are present in a large percentage of the autopsies of the newly born, and may lead to misapprehension.

Acute endocarditis may affect the endocardium in any part. When it is present upon the membrane lining the cavities of the heart, it is commonly termed parietal, when—as occurs in the overwhelming proportion of cases—it



FIG. 128.—Acute endocarditis of the aortic valve.

attacks the valves, it is termed valvular, endocarditis. The part of the valves almost invariably affected is the surface exposed to the blood current; that is to say, the auricular surface of the venous valves, and the ventricular surface of the arterial. The lesions are situated not quite at the edges of the valves, but, as was first noticed by Hodgkin, at that part which comes strongly in contact with another cusp, as is

shown in Fig. 128. It cannot be doubted that this is the effect of strain.

A large series of lesions may be established in endocarditis, which obviously depend upon the severity of the process, the extent of the reaction, and the duration of the disease. It is impossible to draw any definite line separating these lesions from one another. They form a chain of which the ends, although extremely diverse, are yet united by links presenting a perfect gradation. Such changes as thickening, roughness, granulations, abrasions, and ulcerations may be seen, and the reaction processes may lead to union of the affected cusps producing still further alterations.

*Acute Endocarditis.*—The earliest stage in the development of acute endocarditis is a slight dulness of the membrane from accumulation of the products of irritation, along with cloudy swelling of the tissues. As these changes increase, a thickening of the part occurs, and a distinct prominence is produced, which may present a colourless, grayish, or greenish aspect. This can sometimes be removed by gentle pressure, and if it can be so removed, it leaves a distinct abrasion of the surface, which is sometimes distinctly reddish in colour. Smaller projections arise from the free surface of the little prominence, and give rise to a somewhat cauliflower-like body. The process may become less acute, and the mass in this case assumes a firmer aspect, and a harder consistence. On the other hand, in the most serious cases the thickened portion gives way and produces an ulcer, or, after vegetations have arisen, a destructive process may step in, and the vegetations be thrown off. The necrotic process may be so extensive as to produce perforation of a valve, or even of the heart itself, with the separation of large fragments, and the total disorganisation of the valvular mechanism.

The microscopic examination of acute endocarditis in the earliest stage shows the presence of numerous small round cells, arising from proliferation of the connective tissue corpuscles. These are at first aggregated in a mass, but soon stretch in every direction. These small cells are observed in the fibrous layer beneath the endothelium, but they extend into the areolar tissue next to the myocardium, and even invade



the intermuscular septum of the myocardium itself. The endothelium covering the affected part gives way, so that the small cell infiltration projects freely, and deposits of fibrin in wavy masses are seen upon the surface. In the interstices between the layers of fibrin, as well as upon the free surface, are numerous masses of organisms, as may be seen in Fig. 129, and Fig. 130. If the process goes on without any serious necrotic change, the mass finally becomes fibrous, but it may be modified at any stage, and there may be breaking down of the newly formed tissue with ulceration and its sequels.

The illustrations give the microscopic appearances from a section of the wall of the left auricle in a case of acute, following chronic, endocarditis, and accompanied by multiple embolism of the kidney. Large masses of micrococci

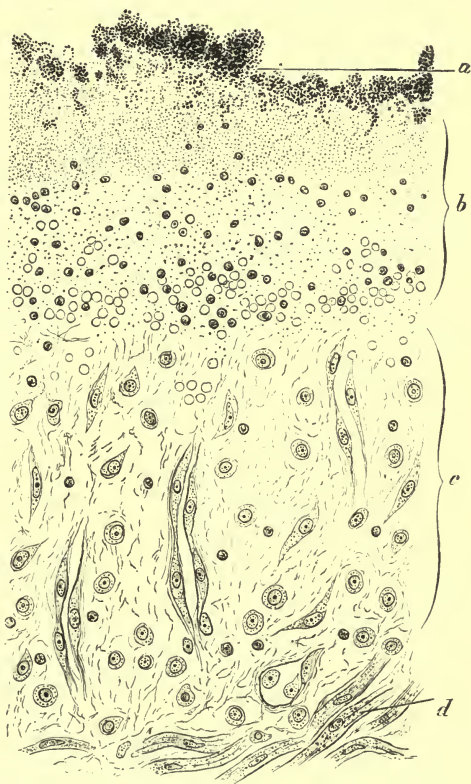


FIG. 129.—Section of the left auricle from a patch of acute endocarditis following a chronic lesion,  $\times 200$ . *a*, Layer of micrococci; *b*, layer of necrosed tissue with leucocytes and haemocytes; *c*, layer of organising tissue, with young connective tissue cells, and newly formed vessels; *d*, layer of muscle cells nearly healthy.

(*staphylococcus pyogenes aureus*) are seen upon the surface. A layer of necrosed tissue, showing numerous leucocytes and red blood corpuscles, is immediately underneath it, while still more deeply is situated a layer of organising tissue with young connective tissue cells and new blood vessels.

Even in this instance, which may be regarded as belonging to the type usually termed malignant, there is a very considerable effort on the part of the organism to protect the deeper tissues from the effects of the disease.

In the illustrations the small cell infiltration of the endocardium may be distinctly recognised, and the deposit of fibrin undergoing organisation is very obvious, while a recent deposit of fibrin is to be seen upon the surface of the vegetation.

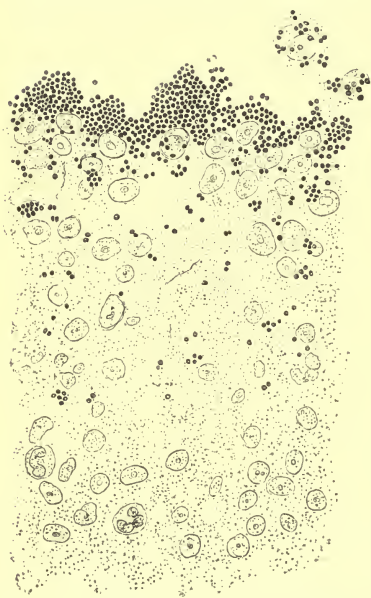


FIG. 130.—The same section of acute endocarditis,  $\times 1000$ , showing a layer of staphylococcus pyogenes aureus on an ulcerated surface, with cocci scattered through the deeper necrosed tissue.

During any part of the disease embolism may take place, giving rise to simple infarction or pyæmic abscess in other parts. When the left side of the heart is affected the organs most likely to suffer are in order of frequency the kidneys, the spleen, the brain, and the liver. In rare cases emboli may block the coronary arteries. In endocarditis affecting the right side of the heart, the embolic processes implicate the lungs. Connected with this aspect of the subject is the fact that when there have been lesions of the endarterium, vegetations are apt to arise in the diseased tissues.

#### *Subacute Endocarditis.*—

The tendency of the disease in subacute endocarditis is much more conservative than in the acute form, and the resulting lesions are accordingly much more vegetative or verrucose. On account of the greater tendency to the formation of fibrous tissue, the granulations are more permanent, and there are also at once more adhesions between the cusps, and less loss of tissue.

The appearances presented by the diseased structures are

thickenings of the endocardium with firm vegetations. Adhesions of the cusps to one another are also common.

Microscopic examination reveals an infiltration of the endocardium with small round cells and fibrinous granulations, partly formed from the endocardium, but partly also having origin in a deposition of fibrin from the blood. These appearances are seen in Fig. 131.

The presence of micro-organisms can often be determined



FIG. 131.—Section of subacute endocarditis of the auricular surface of a mitral valve,  $\times 20$ . *a*, Fibrinous coagulum; *b*, recent fibrin on the surface of the vegetation; *c*, endocardium infiltrated with small round cells; *d*, wall of the valve.

in such cases of subacute endocarditis. The bacteria found in them are identical with those referred to in connection with the acute variety.

*Chronic Endocarditis.*—In this special form of the disease the reparative and conservative processes are still more in the ascendant; it therefore happens that the presence of firm vegetations, resistant thickenings, and rigid union of the cusps is particularly characteristic of this variety. The deposition of lime salts is very common. The lesions constitute one large

group of the chronic valvular lesions to be separately discussed.

The appearances found in chronic endocarditis are, therefore, various degrees of deformity of the valves, and deposits upon the endocardium elsewhere. These lesions often assume an intensely firm character from calcification. It was long ago pointed out by Hodgkin that the masses connected with the valves have a tendency by friction to set up changes in near structures.

Microscopic examination of sections of chronic endocarditis shows dense fibrous tissue, usually arranged in strata, often associated with deposits of freshly formed fibrin upon the surface. In this fibrin various organisms are found. There may also be masses of a calcareous nature amongst the fibrous tissue. The ordinary structure is shown in Fig. 132.

The statistics of Sperling from the Berlin Pathological Institute, in regard to the incidence of endocarditis and the occurrence of embolic

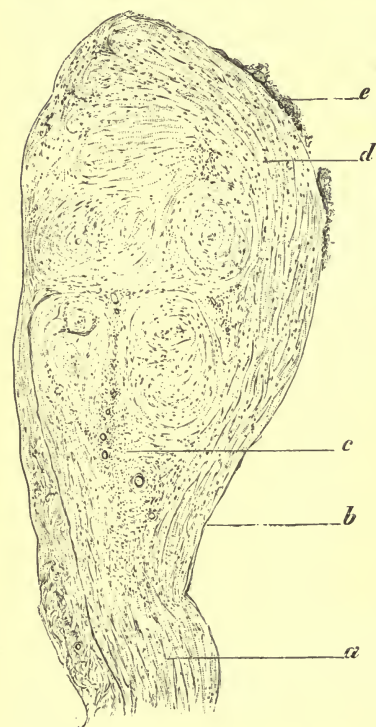


FIG. 132.—Section of chronic endocarditis of the aortic valve,  $\times 15$ . *a*, Healthy part of the valve; *b*, ventricular surface; *c*, small cell proliferation; *d*, dense fibrous tissue arranged in layers; *e*, recent fibrin deposited on the thickened valve.

sequels, are of so much importance that they may well be given here.



300 Cases of Endocarditis in the years 1868-70.

268 cases = 89 per cent, left side of the heart affected.

3 " = 1 " right " "  
29 " = 10 " both sides "

Total 300

*Affection of one Valve only.*

200 Cases = 66·7 per cent.

Of which :

Mitral valve . only, 157 cases = 78·5 per cent.  
Aortic valves . " 40 " = 20·0 "  
Tricuspid valve . " 3 " = 1·5 "  
Pulmonary valves " 0 " = 0 "

*Combined Valvular Lesions.*

100 Cases = 33·3 per cent.

Of which :

Mitral and aortic . . . 71 cases = 71 per cent.  
" tricuspid . . . 9 " = 9 "  
" pulmonary . . . 2 " = 2 "  
Aortic and pulmonary . . . 1 " = 1 "  
" tricuspid . . . 0 " = 0 "  
Mitral, aortic, and tricuspid . 16 " = 16 "  
" " pulmonary . 0 " = 0 "  
Tricuspid, pulmonary, and mitral 0 " = 0 "  
" " aortic 0 " = 0 "  
All four valves . . . 1 " = 1 "

*Embolism.*

84 Cases = 28 per cent.

Of these 76 occurred in connection with the left side, and  
8 with the right side of the heart.

Kidney . . . . . 57 cases.  
Spleen . . . . . 39 "  
Brain . . . . . 15 "  
Digestive organs . . . . . 5 "  
Skin . . . . . 4 "

If in the above table any of the combinations of valvular lesions are wanting, it must not be supposed that they are never observed ; the figures occurred in the course of the years in which the statistics were compiled, and only refer to them.

SYMPTOMATOLOGY.—Endocarditis produces an immense variety of symptoms. No two examples of the disease are ever exactly alike, yet the different clinical features form a continuous series. It is therefore by no means an easy task to draw distinctions between different varieties, and it is impossible to read without a certain degree of admiration some of the systematic descriptions of the different forms of the disease in which, with a cheerful confidence, they are separated into distinct types.

From the clinical standpoint it seems possible to take as the rallying point of certain forms of the disease three great types, using this phrase with the reservations already mentioned, these being: first, the acute, malignant, or ulcerative; second, the subacute, verrucose, or vegetative; third, the chronic, sclerotic, or contracting.

*Acute Endocarditis.*—Cases falling under this head show very different symptoms, yet they are marked by one or other of two diverse tendencies. There is, in the first place, a class in which the symptoms are characterised by typhoid characters. The temperature in such cases is high and sustained; the pulse is empty and compressible, usually frequent, and often dicrotic, or even hyperdicrotic. It is at first usually regular, but in the later stages it may be extremely irregular. The respiration is shallow and hurried. There are profuse perspirations. The tongue is dry throughout, having often, in the earlier period of the disease, a brown fur in the centre bordered by red edges; it may, however, be cracked or fissured. As the disease progresses there is often meteorism and sometimes diarrhoea, occasionally melæna. Choleric form diarrhoea was described by Trousseau. The spleen is usually enlarged, the urine often contains albumin, and sometimes blood. A lethargic or soporose condition is common, tending in some instances to pass into coma, and characterised in others by low muttering delirium. There are in some cases rosy papules upon the skin, and sudamina are extremely common. Purpuric appearances have been seen, and Claisse found the same organisms in them as on the valves.

Patients who present this particular group of symptoms have, in many instances, extremely few physical signs connected

with the heart, except weakness of the cardiac impulse and sounds. If the affection, however, is not rapidly fatal, evidence of cardiac implication shows itself by an enlargement of the area of dulness, more particularly to the right, along with a progressive diminution in the intensity of the first sound, and the development of murmurs. These murmurs are for the most part confined to the mitral and tricuspid areas, and they are systolic in rhythm, but they may be presystolic in these situations, while in the aortic and pulmonary areas systolic, and even diastolic, murmurs may be heard.

In consequence of embolic processes many appearances arise, connected not only with internal organs but with superficial parts of the body; in fact, owing to the severity of the general symptoms, the former are apt to be overlooked, and the latter therefore assume greater importance. Upon the skin there may be papules which, from their pale or yellow centre, show their embolic origin; these sometimes become vesicular, in a herpetic or pemphigoid manner, or may suppurate, and give rise to pustules. These appearances are often accompanied by erythematous blushing, or subcutaneous extravasation of blood. The mucous membrane of the lips, gums, and tongue may have small hæmorrhages, which show their embolic origin by a pale centre. In one such instance, Eichhorst mentions that he has seen subsequent ulceration of the mucous membrane. The conjunctiva is often bloodshot from embolism of its blood vessels, and sometimes, as was pointed out by Litten, on ophthalmoscopic examination there are hæmorrhages in the retina, which, by means of the pale central portion, reveal their origin. Occasionally in the less rapid cases there is necrosis of the retina.

The course of acute endocarditis in this typhoid form is usually rapid. In a case described by Eberth, death occurred on the second day of the disease, and in one mentioned by Eichhorst a fatal result occurred on the fourth day. It may, however, happen that the affection passes into a somewhat less acute form, and may even assume the features of chronic endocarditis. Death most commonly occurs on account of acute general infection, but sometimes from heart failure, in consequence of paralytic myocarditis; it may, however, be

through implication of the nervous system by embolism, or by complication connected with the thoracic viscera.

The other form in which the disease shows itself is characterised by its septicæmic features, commencing with severe and usually repeated rigors. It presents for the most part the characters of an intermittent pyrexia. The temperature is extremely irregular and intermittent. The pulse rate undergoes great fluctuations, and the febrile periods very commonly terminate by means of profuse perspirations. There is usually complete anorexia, and the tongue is heavily furred. Various digestive disturbances, such as tympanites and diarrhœa, occur. The spleen is very commonly enlarged, and during the attacks of pyrexia there is often delirium. The most careful examination of the circulatory organs often fails to give any definite indication of a lesion. The pulse is as a rule empty and compressible, often fully dicrotic, and sometimes extremely irregular. The cardiac impulse is usually feeble, and the area of dulness is often enlarged, especially towards the right. Systolic murmurs in the mitral and tricuspid regions, as well as near the pulmonary area, along with accentuation of the second pulmonary sound, are common, but all these symptoms are no more than may be found in any acute pyrexia. Obstructive murmurs may develop themselves while the patient is under observation, and in this way give valuable evidence with regard to the lesion which is present.

Embolie symptoms are often present. The sudden onset of pain in the back, followed by hæmaturia, may show that embolism of the kidney has taken place. A rapidly occurring hemiplegia may indicate cerebral embolism. The occurrence of peritonitis and of pleurisy can sometimes be traced to the same process, affecting the serous membranes; and various affections of the skin and mucous membranes, such as have been referred to previously, may own the same origin.

This septicæmic form of acute endocarditis is sometimes specially characterised by the cerebral symptoms to which Osler has particularly called attention; it is then frequently associated with meningitis, and it may be noted that Netter has detected pneumococci in the meningeal exudation.

It cannot be too strongly insisted upon that a great many



cases of this affection run a perfectly latent course, and only reveal themselves by a sudden disturbance of the circulatory system, through changes taking place in the heart, or embolic processes occurring in other organs. An irregular and intermittent pyrexia may give rise to considerable difficulty in diagnosis, when the sudden development of a previously absent valvular lesion, or the onset of cardiac failure, may show that a profound change is taking place in the heart. There may be, on the other hand, along with a similar febrile type, characteristic appearances denoting the incidence of embolism. Forms of endocarditis, such as have been here mentioned, often run a prolonged course. The disease may continue in its acute form for weeks, and cause death by asthenia, or by one of the many complications to which it is liable; it may pass into a less acute form with occasional exacerbations, and continue for months, or it may even assume a still more chronic form. The end, however, has, until lately, been almost invariably fatal.

*Subacute Endocarditis.*—Subacute, verrucose, or vegetative endocarditis is frequently unattended by any general symptoms. When it occurs as a complication of some general disease, as in acute rheumatism, it frequently does not even cause any elevation in the temperature, and but little alteration in the pulse or respiration. Even in those instances in which subacute endocarditis has taken its origin in some chronic disease, as in the case of chronic nephritis, the cardiac affection can sometimes only be detected by careful physical examination of the heart. It may, on the other hand, be attended by a rise of temperature, with accelerated pulse and hurried breathing.

In some cases there are subjective sensations connected with the heart, such as uneasiness, even amounting occasionally to pain, and palpitation, which is sometimes almost entirely a subjective symptom. Syncopal tendencies may be present.

The pulse may undergo no alteration, but it is more common to find that it becomes less full and more compressible than in health, with increased frequency, and sometimes irregularity. In some instances pulsations may be observed in the veins of the neck. The apex beat becomes feeble, although from the excitement of the heart, its sharp impulse is apt to be mistaken for an exaggerated beat. It is

often further to the left than in health, and it may be attended by a thrill. The cardiac area of dulness is very commonly increased, particularly to the right. On auscultation there are great possibilities of error. In all febrile conditions, such as so often lead to endocarditis, the heart is apt to undergo dilatation, and not only is there a change in the cardiac sounds but systolic murmurs are extremely common at the auriculo-ventricular orifices, along with accentuation of the pulmonary second sound. Such murmurs may obviously be caused by changes in the cardiac walls, or in the papillary muscles, and therefore have no necessary connection with endocarditis; as has already been remarked, reliance upon these murmurs as an indication of endocarditis has rendered most clinical statistics connected with the subject open to the gravest suspicion. It cannot be urged too strongly that the only absolute diagnostic means consists in the appearance of murmurs of obstruction at one or other of the orifices.

Sibson regards the existence of a mitral systolic murmur, in a first attack of acute rheumatism, as a direct sign of endocarditis. This statement is much too absolute, and is not borne out by the after history of such cases. It seems to me that the views of Sansom on this subject are extremely just. He considers that great weight should be allowed to the date of the development of the murmur. Those cases presenting the evidence of mitral incompetence at an early period of the febrile attack are probably endocarditic in origin, since there has not been time for the development of regurgitation by means of muscular relaxation; in others, with a late appearance of such features, the probability is that the valves are unaffected. Sibson is beyond all question justified by clinical and pathological experience in believing that systolic tricuspid murmurs, in febrile affections liable to produce endocarditis, are due to the "safety-valve action" of the right venous cusps. Surely he goes too far, however, when he concludes that systolic tricuspid murmurs in such cases are the result of left-sided endocarditis. The determination of the probable condition in any given case can only be attained by carefully weighing the whole evidence available, and the possibility that murmurs of incompetence

at the venous valves may be due to acute myocarditis must not be overlooked. This latter suggestion will be again referred to in another section.

If a patient, while under observation, develops a pre-systolic murmur in the mitral area, or a systolic murmur in the aortic area, there can be no doubt that endocarditic lesions have made their appearance on the cusps of the respective valves. The evolution of an aortic diastolic murmur is often to be traced in endocarditis, and it always means that part of the valvular apparatus has, in consequence of the local lesion, been retracted, or that in consequence of vegetations the cusps are prevented from fully closing the orifice, or that part has been swept away in the blood current. It is not uncommon to find the development of a pulmonary diastolic murmur while endocarditic processes are going on.

Breathlessness, cough, and other pulmonary symptoms may arise in consequence of implication of the lungs from weakness of the heart. Albumin, blood, and casts may be present from the secondary effects of cardiac failure on the kidneys. Headache, restlessness, and sleeplessness may in the same way be produced by the failure of the circulation to supply the brain, as well as by the influence of the febrile process.

As in the case of the more serious malignant forms of endocarditis, the variety now under consideration frequently leads to embolic processes. Sudden pain in the loins, with rigors, followed by hæmaturia, may give evidence of renal embolism. Pain in the left hypochondrium, also frequently attended by shivering, and followed by an enlargement of the area of splenic dulness, may be significant of splenic embolism. The sudden development of paralytic symptoms may be traced to cerebral embolism. Pain, with hæmorrhage from internal mucous membranes, may point to the probability of embolism of some part of the alimentary tract, and local absence of the pulse with cold surfaces, lancinating pains, and paralytic symptoms, may result from embolism of an artery of a limb, leading, in the case of an important vessel, to gangrene. Sudden death from cardiac failure may be the consequence of coronary embolism.

Erythema nodosum is not infrequent as an accompaniment of subacute endocarditis. It has been held to be a result of embolism, but as it is in every instance associated with rheumatism, it is much more probable that, like chorea, it has its origin in the local action of some as yet unknown toxic body, produced by the action of an organism, or of some poisonous substance from perverted chemical activity.

Other complications may arise in the course of subacute endocarditis. It is extremely probable that in a large proportion of cases myocarditis arises by direct extension from the diseased endocardium, and, by its paralysing effects, is the cause of the weak, irregular, and frequent pulse, the feeble heart sounds, the dyspnoea and syncopal seizures so often observed. It is equally probable that the pericarditis, which can often be made out by physical examination, is likewise a result of direct extension. The possibility, however, must not be overlooked that all these cardiac lesions may have their origin in the primary affection from the same cause. Acute aortitis has been described as a sequel of the disease more especially by Petit. It may be suspected, as will afterwards be more particularly mentioned, by pain, radiating towards the shoulder, and by a dulness of the second sound in the aortic area. Other acute complications, such as pneumonia and pleurisy, are usually the result of the primary cause.

The course of subacute endocarditis is, as a general rule, more lengthy than even that of the intermittent type of the acute form of the disease, and its termination is commonly gradual recovery of the patient with a chronic valvular lesion as the local result. It is held by several authors, as has already been mentioned, that absolute recovery may ensue, but to my mind the evidence in favour of such a view is open to question.

*Chronic Endocarditis.*—Chronic endocarditis is unattended by any general symptoms, apart from those which are a result of the circulatory disturbances having their origin in it. Pyrexia is only present if an acute attack of endocarditis is grafted upon the chronic process, or if some complication arises in consequence of the liability to acute changes in parts affected by venous stasis. The temperature is not,



therefore, necessarily altered in chronic endocarditis, and the changes of pulse and respiration found in pyrexia are absent.

The local symptoms and physical signs of chronic endocarditis are solely connected with the valvular lesions which it produces, and the circulatory disturbances resulting therefrom. They will therefore be considered under the head of chronic valvular lesions.

DIAGNOSIS.—The recognition of the acute form of endocarditis is attended by very considerable difficulty. In addition to the care necessary to determine the presence of endocardial disease, along with septic infection, there are certain other diseases for which it is apt to be mistaken, more particularly enteric fever, acute tuberculosis, and septicæmia.

Septicæmia is above all others the disease for which acute endocarditis is apt to be mistaken. Little wonder need be caused by this, seeing that most cases of acute endocarditis are of septicæmic origin. In all septicæmic affections, whether complicated by the existence of an endocardial lesion or not, there are characteristic features of pulse, skin, and temperature. The sole distinction that can be drawn between cases with and without endocarditis must be deduced from the physical examination of the heart.

Acute tuberculosis is less likely to be confounded with endocarditis. Not only are periodic fluctuations of pulse and temperature, as well as profuse nocturnal perspirations, more regular in their appearance, but there is also much more rapid wasting. There are often, moreover, definite clinical features of a local implication, *e.g.* of the lungs, or of the brain. In the event of any pulmonary symptoms there may be some evidence of an apical change, and it may even be possible to discover the tubercular bacillus if there be any expectoration. In almost all cases, lastly, of general tuberculosis, the heart gives absolutely negative evidence on physical examination.

Enteric fever may exist in such obscure forms as to be readily mistaken for other affections such as endocarditis. Every clinical feature of diagnostic value, such as the rash or

the stools, may be absent, and much difficulty in diagnosis is thereby introduced. Nevertheless, even in such doubtful cases the regularity and infrequency of the pulse, and the characteristic temperature curve, may be useful in distinguishing the disease from endocarditis, even when cardiac symptoms and signs are present. It need hardly be added that at the present time, any doubt would at once be submitted to the arbitrament of bacteriological investigation; that the beautiful phenomena produced by the blood when added to a culture of enteric bacilli, as discovered by Gruber and Widal, would at once be sought for.

The physical signs of auriculo-ventricular incompetence may obviously arise either from endocarditis or myocarditis. Both these affections are common in acute febrile diseases; in many cases, no doubt, the myocarditis is the result of, or at least is caused by extension from, an affection in the first instance of the endocardium; many instances of myocarditis, notwithstanding, are undoubtedly produced by the action of toxins directly on the heart-muscle. In such instances it may be absolutely impossible to determine whether the heart muscle or the lining membrane is affected, in the absence of physical signs of obstruction at an orifice.

The recognition of subacute endocarditis is not attended by so much difficulty, and it only requires care on the part of the observer during the course of such affections as are likely to be complicated by its presence. As in the case of pericarditis, so here also endocarditis must be sedulously watched for during the course of acute rheumatism, acute pneumonia, and all other conditions liable to be complicated by the development of endocardial affections. The chief difficulty in the diagnosis of subacute endocarditis lies in the possibility of mistaking a simple dilatation, produced by pyrexia, for an endocarditis, and it cannot be too strongly insisted upon that systolic mitral and tricuspid murmurs are in themselves apt to be misleading. It is only in the presence of murmurs of obstruction or of a diastolic aortic murmur that it is possible to attain certainty. Murmurs of regurgitation, nevertheless, when developed early in the course of the acute disease, are, as has previously been stated, of more value as diagnostic

indications. Much, however, depends on the severity of the initial disease.

A prolongation and muffling of the first sound has been regarded by many authors as the most frequent early sign of endocarditis. Gull and Sutton, Sibson, and Sansom all agree on this point. The reasons adduced previously as to the doubtful value as evidence of a murmur of regurgitation apply here also with almost greater force.

The diagnosis of chronic endocarditis is practically that of valvular disease, and the only difficulty is to distinguish between the probability of a valvular lesion being the result of endocarditis or degeneration. This subject belongs naturally to the chapter which is devoted to valvular disease.

PROGNOSIS.—The outlook in endocarditis may be regarded as steadily brightening on passing from the acute, through the subacute, to the chronic form of disease. The prognosis in the acute variety is still gloomy, but with the light thrown on the disease by modern research, and the aid rendered by recent investigations, there are grounds for hope that the disease will shortly be deprived of its former hopeless prospect. Subacute endocarditis may be regarded in the main as tending towards a favourable immediate termination, yet it is sometimes directly fatal. Chronic endocarditis being, from the standpoint of practical medicine, simply a part of chronic valvular disease, need not be discussed here.

In forming a prognosis in any case of endocarditis several facts must be steadily kept in view. Among these may be mentioned the character of the primary affection causing the lesion. In all instances in which the affection is simply part of a general infection much depends upon its severity. Cases of septicæmia, for example, are so frequently fatal in themselves that the cardiac complication is but an episode. In the course of enteric fever, on the other hand, the original factor is not so serious. Notwithstanding such a consideration, however, it must be remembered that, in consequence of mixed infection, a fatal attack of endocarditis may be grafted upon a disease in itself of no great severity. The state of the heart itself, as well as of the circulation in general, must therefore receive the greatest consideration,

and such symptoms as point to cardiac failure must be allowed great weight in coming to a conclusion.

TREATMENT.—The management of endocarditis provides a wide field for the exercise of practical medicine. The extensive range of primary causes and the variability of clinical features render it somewhat difficult to lay down definite rules, but general principles of treatment may easily be formulated.

As regards, in the first place, *acute endocarditis*, it will naturally occur to the physician in charge of any general or local disease liable to this cardiac complication to watch for symptoms warning him of its approach, and, if there be any suspicion that it is imminent, resource will be had to prophylactic measures.

Until quite recently the only methods available have been sought in antiseptic remedies. Now, however, the knowledge of serum therapeutics has placed in the hands of the physician a means of combating the disease directly. The whole subject is, at present, however, in an experimental phase, but the results obtained thus far are full of encouragement.

Seeing that the most common agents in acute endocarditis, whether it arises in consequence of a single or mixed affection of the endocardium, are pyogenic organisms, the particular form of serum to be employed will necessarily be anti-streptococcic serum. But, when other forms of infection are determined or suspected, the appropriate serum for each case will be resorted to. There need be no hesitation in the use of these serums. Frequent use of them in many different conditions has convinced me that they are absolutely harmless, and it is, therefore, our bounden duty to have resort to such measures in order to cope with these forms of endocarditis which have hitherto been regarded as hopeless. It need hardly be added that, from the point of view of prophylaxis, careful attention must be given to the general conditions by which the patient is surrounded.

Cases in which acute endocarditis has already occurred do not afford so much possibility of relief; yet even in them the disease cannot be regarded as beyond hope. Absolute rest, free ventilation so as to ensure oxygenation of the blood,



warmth of the extremities, in order to ease the peripheral circulation, and appropriate food, must be ensured. If there be any tendency to cardiac collapse, the employment of alcoholic and ammoniacal stimulants, along with cardiac and nervine tonics, will be employed. Such antiseptic drugs as quinine, salol, naphthol, and the sulpho-carbolates have been largely employed in the past, but now various blood serums give much more hope of benefit. Sainsbury and other writers have placed on record instances successfully treated in this way, and there can be no doubt of the efficacy of antistreptococcic serum. It must always be a matter of doubt whether, in cases in which the affection has got a considerable start of treatment, the inoculation will be able to overtake and antagonise the disease process; but the results of several cases which have been recorded are sufficient to render it probable that this can be done. In one case, seen by me with Dr. Boyd at the Deaconess Hospital, which will soon be published, most excellent results followed the use of the serum. Of anti-streptococcic serum the first dose should not exceed 5 c.c., but the quantity may rapidly be increased to 20 c.c. One injection should be given every day, with rigid aseptic precautions. At the same time the action of the heart is to be supported by the use of strophanthus or digitalis, along with stimulant remedies.

It is doubtful if any local measures are of the least utility in this disease. The employment of ice to the præcordia has been advocated, but is apt to be depressant to an already enfeebled circulation. Warmth certainly is of more value. Counter-irritation by mustard, or even by blisters, has also been advocated, but there is a want of common sense in the use of such agents when a general infection is in process. The same may also be said of the extraction of blood by means of leeches.

Turning in the next place to *subacute endocarditis*, the first question which will naturally arise is whether, in cases liable to be complicated by its presence, any prophylactic measures may be adopted with fair prospect of success. As any acute process that may be present will doubtless be under treatment of an appropriate kind, the measures already employed

must be diligently persevered with. Since the overwhelming proportion of cases occurs in the course of acute rheumatism, the use of some member of the salicyl series of drugs will no doubt be the chief element of the treatment, and this must be followed out. But certain other measures seem also to be beneficial. The addition of iodide of potassium to the specific remedies employed, and the systematic use of counter-irritation by blistering the præcordia, afford the best promise of relief. Caton has more especially advocated the latter method, and recommends a succession of small blisters. From his results it cannot be doubted that such treatment does lessen the liability to endocardial complications. The explanation of the action is not easy, but it is probable that the effect upon the peripheral nerves is carried by way of the spinal segments, and their connections with the sympathetic ganglia and nerves, to the heart, where an influence is exerted on the blood vessels of the endocardium.

In the case of other general diseases, whether constitutional or specific, the appropriate treatment for them should also be carried out. When there is any danger of an endocardial complication in acute gout, the special remedies—colchicum and alkalies—are to be pushed. If there is any suspicion of endocardial implication in such an affection as scarlet fever, for which, so far, no known specific remedy exists, the general treatment must be sedulously carried out. In all such cases the use of counter-irritation is to be strongly recommended. Inasmuch as many of these general diseases are complicated by mixed infection, through the addition of pyogenic organisms, an effort should be made to detect such microbes in the blood, and, in the event of a positive result, antitoxic inoculation should be adopted.

When the existence of subacute endocarditis has been determined, the question is no longer one of prophylaxis, but becomes one of remedial treatment. The lines upon which the primary disease has been managed must still be carried out, and such additional measures adopted as are demanded by the new aspect of the case. The necessity for absolute rest, sufficient slumber, abundant air, and suitable clothing need scarcely be insisted upon, but a word

may be specially added in regard to food and drink. Food should be administered every two hours. During the early stages of the affection it should consist solely of milk; this may be diluted with abundant pure water, which may be hot or cold according to the wish of the patient, unless there be any tendency to collapse, when warm fluids are best. The addition of alkalies certainly seems to be useful, and the water may be aerated if it does not give rise to gastro-enteric distension. The action of the bowels should be watched, and gentle saline aperients given if necessary—Rochelle salt or sulphate of sodium acting very well.

In many cases no further treatment is required, except the employment of external measures by counter-irritation. This may be carried out as above mentioned. In some cases when pain over the præcordia is a prominent feature, leeches may be applied, or hot fomentations or poultices used; it is certainly good practice, moreover, in such instances to add the internal administration of anodynes. Of all these, the best for this purpose is morphine.

The physician must be on his guard against cardiac failure. Paralytic myocarditis may ensue with alarming rapidity, and suddenly end the scene. Against it the cardiac tonics, digitalis and strophanthus, along with free stimulants, are the best antagonists; of all means, Niemeyer's pill of quinine, opium, and digitalis seems to me to be most useful. It may be necessary to have recourse in certain cases to the use of strychnine hypodermically, in order to sustain the nervous centres, and inhalations of oxygen should also be resorted to if the respiration becomes embarrassed.

After the more acute stage has passed off, the food may be varied by the addition of eggs, fish, and chicken, gradually added to until ordinary diet is reached. Absolute rest must for a considerable time be enjoined. Iron, arsenic, quinine, and strychnine may be given in varying combinations, but whatsoever of such a nature is administered, it seems to me of even greater use to persist in the use of iodide of potassium for a prolonged period.

It is unnecessary in this section to deal with the treatment of *chronic endocarditis*; the management of its different forms

will fitly fall under the diseases of the orifices and valves to which it so largely contributes.

The following cases are illustrative of some of the points which have been dealt with.

CASE 13. *Acute Endocarditis with Intermittent Course.*—A. L., aged sixteen, domestic servant, was admitted to Ward 25 of the Royal Infirmary, 19th October 1891, suffering from acute rheumatism.

There were no hereditary tendencies of any importance as regards the disease from which the patient was suffering. Her previous health had always been excellent. She had from early years been accustomed to hard work, but her surroundings had always been satisfactory.

Eleven days before her admission the patient had developed a severe attack of acute rheumatism, which, after a few days, led to cardiac complications, when she was recommended to the Infirmary.

On admission the patient was very anæmic and extremely weak. Her tongue was heavily coated with a white fur, and there was complete anorexia. There was no alteration in the condition of the digestive viscera on physical examination. The spleen was not enlarged. The pulse was empty and compressible, but perfectly regular, varying between 100 and 120, with a small weak pulse wave.

There were slight venous undulations in the neck. The apex beat was in the fifth interspace, four inches from mid-sternum. It was so feeble as to be difficult of detection. There was no thrill over any part of the præcordia. The upper border of the heart reached the third rib. The right border was one inch and a half to the right of the mid-sternal line; and the left extended four inches and a half to the left of mid-sternum. Faint murmurs, presystolic, systolic, and diastolic, could be heard in the mitral area. A tricuspid systolic murmur was distinctly audible. In the aortic area a distinct systolic murmur and a very faint diastolic murmur were heard, but sometimes the diastolic murmur was inaudible, and reduplication of the second sound was present. A loud systolic murmur was present in the pulmonary area, but this could not be differentiated from the tricuspid murmur.

Cerebral breathing was present. There were no urinary symptoms, but the nervous system was obviously depressed, for the patient manifested a lethargic condition with a stolid aspect and dull senses.

It was quite clear that in this case, in addition to a febrile dilatation of the heart, as evidenced by the systolic mitral and tricuspid murmurs, there was also some obstruction at the mitral and aortic orifices, no doubt produced by recent vegetations, and it could not be doubted that there was either some slight destruction or retraction of one or other of the aortic cusps, as evidenced by the diastolic aortic murmur. The escape, however, was but slight, and the cessation at times of the diastolic murmur showed that the structural change could not be extensive.

The patient was kept absolutely at rest and placed upon milk diet. On account of the depressed condition of the nervous system, she received



The case may be held to furnish a good example of the intermittent type of acute endocarditis.

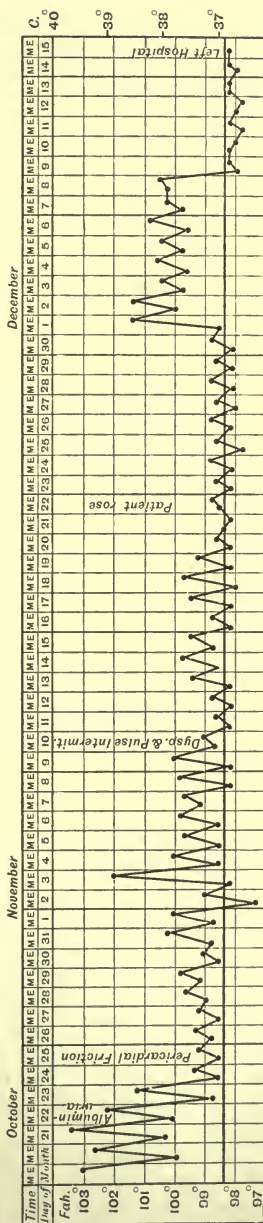


FIG. 133.—Temperature chart from Case 13.

CASE 14. *Acute Intermittent Endocarditis*.—A. S., aged 22, factory girl, was sent to see me on 10th February 1896, in the out-patient department of the Royal Infirmary, by my colleague, Dr. Maddox. She had for two or three weeks been attending the eye department, and Dr. Maddox desired a report on the state of the heart.

The patient's father and mother were alive and had always been well. Her only brother had died as a baby, two sisters also died in infancy, and one, after miscarriage, in child-birth. Her two remaining sisters were in good health. So far as could be ascertained, there were no family tendencies towards circulatory disease.

The patient's social conditions were not unfavourable, and her previous health had always been perfectly good, with the exception of an attack of measles. During the month of November of the previous year the patient somewhat suddenly began to suffer from breathlessness, and it was noticed that her complexion became pallid. Amenorrhœa had been present since the New Year. She continued her work, nevertheless, until, about a month before she was sent to me, she found that the sight of her right eye was affected, and she sought advice in the eye department of the Infirmary.

The patient was 5 ft. 1 in. in height and weighed 7 st. 2 lb. She was slenderly formed, but well nourished, with an extremely pale complexion and bloodless lips and gums. The temperature was normal. There were no symptoms connected with the alimentary system. The spleen and lymphatic glands showed no abnormality.

The pulse was 94, the vessel was empty, the blood pressure low, the pulse wave large, bounding, and collapsing, but perfectly regular. On causing a reddened patch on the forehead, distinct capillary pulsation was visible. The fingers were clubbed and the nails arched. They also showed a capillary pulse. On examining the chest, it was seen that there was very distinct pulsation in the carotid arteries, but there was no other abnormality on inspection. On palpation, the apex beat, which was forcible, was found in the fifth intercostal space, four inches from mid-sternum. Percussion showed that the upper border of the heart was at the third costal cartilage, the right border one inch and three-quarters, and the left five inches from mid-sternum. Auscultation revealed loud systolic and diastolic murmurs, heard over the whole præcordia, indeed throughout the whole chest; the point of maximum intensity of the systolic murmur was at the upper edge of the manubrium sterni, almost in mid-sternum; the diastolic murmur at its point of maximum intensity was at the left edge of the sternum, where it was joined by the third costal cartilage. There were no abnormal sounds except these murmurs over the præcordia. The respiratory and urinary systems gave no sign of disease, and, with the exception of the blindness, there was no nervous symptom.

This case appeared to be probably one of an insidious endocarditis, but the most rigorous examination failed to elicit any definite causation. It was quite obvious that the main valvular affection was connected with the aortic cusps, but on account of the absence of any definite

second sound, and of the localisation of the murmurs, it was impossible to avoid the suspicion that there might also be some affection of the pulmonary valves. This suspicion was undeniably strengthened by the appearance of the patient's fingers, for there was a degree of clubbing and arching difficult to explain in the absence of any symptoms of backward pressure.

The patient was sent back to Dr. Maddox with a report upon her condition, but, as matters did not seem to me to bode well for her future, she was requested to present herself again in a week for further examination.

On her return she was clearly in a worse condition than when seen the week before, and she was therefore recommended to remain in the Infirmary in order to obtain the benefit of further treatment, to which

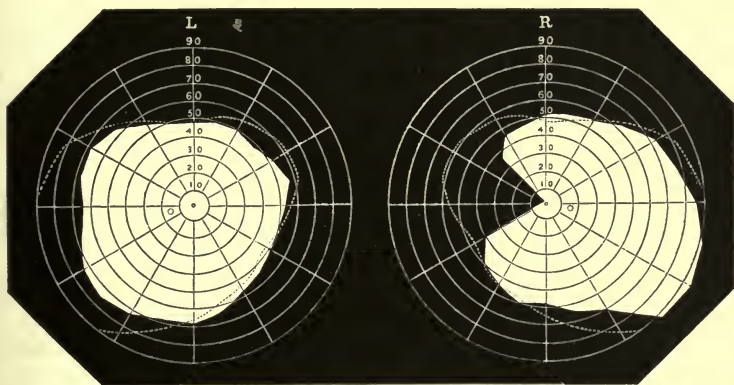


FIG. 134.—Fields of vision in Case 14.

suggestion she gladly consented. She was therefore admitted to Ward 25 on 17th February 1896.

Her residence in the Infirmary allowed of further observations, and the facts which have been already mentioned were more fully determined. In addition to these, however, some other points were ascertained.

The temperature was found to be almost always elevated. It did not rise to any very great extent, but fluctuated for the most part between  $99^{\circ}$  and  $100^{\circ}$ , rising, however, at times to  $103^{\circ}$ , and sinking at other times to  $98^{\circ}$ . The examination of the blood showed 20 per cent. of hæmoglobin, and 2,300,000 red blood corpuscles. The examination of the fields of vision showed that of the left eye to be quite normal, while that of the right eye was greatly restricted in its nasal portion. Ophthalmoscopic examination explained this symptom, for it showed characteristic appearances of embolism. The patient remained under treatment until 23rd April. Her condition fluctuated considerably, but under the influence of salol, iron, and arsenic, with digitalis from time to time, she somewhat improved, and when she left, her temperature was steadier and her general



condition better. On the 8th May she again presented herself with all her symptoms exaggerated, and she was at once admitted to the ward. Examination of the heart showed that there was great enfeeblement. Both in the mitral and tricuspid areas the first sound was very faint; its condition otherwise remained as it was when she was admitted for the first time. She suffered greatly from breathlessness. The temperature on her admission was subnormal. The pulse, however, was 116 and the respirations 36. There was a considerable amount of cough, and a large quantity of frothy sputum, greenish for the most part in colour, but tinged in parts with red. Examination of the lungs revealed some dulness on percussion at the right apex, but the sound elsewhere was unaltered. Auscultation showed that the breathing at the right apex was bronchial, elsewhere it was vesicular. There were crepitations

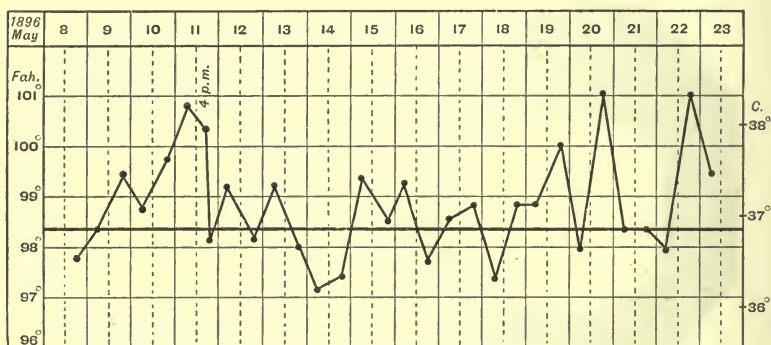


FIG. 135.—Temperature chart from Case 14.

widely scattered throughout both lungs, and the vocal resonance was greatly increased over the right apex, but was normal elsewhere. There was some œdema about the ankles, but there was no albumin in the urine. The temperature oscillated a good deal during the few days immediately succeeding her admission, as is seen in Fig. 135, and she suffered greatly from the respiratory symptoms. On this occasion she was again treated by means of salol. Strophanthus was also exhibited along with aromatic spirits of ammonia, sedatives, and warm applications. Day by day, however, the patient grew steadily worse, and in spite of every method of treatment, she died on 23rd May. A post-mortem examination could not be obtained.

This case presents a considerable number of difficulties, but taking everything into account, the most probable explanation is that of an intermittent acute endocarditis, which might be regarded as of a malignant type.



CASE 15. *Subacute Endocarditis*.—J. W., aged 25, commercial traveller, was seen by me in consultation with Dr. Veitch, 25th March 1897, on account of the consequences of an attack of acute rheumatism.

The patient's family showed some slight tendencies towards rheumatism. Both his parents were alive and healthy, as were also his brothers and sisters, but one of the brothers had suffered from time to time from undeniable rheumatic symptoms. The patient had been married about two years, and one child, a strong healthy boy, had been born of the marriage.

The patient's surroundings and occupation had been perfectly satisfactory. The illness for which he was under treatment had begun about three weeks previously, and had presented every feature of a mild attack of acute rheumatism, the temperature having reached a level of  $101^{\circ}$ , while the disease had attacked the knees.

On examination the patient was found to be somewhat emaciated, with a large bright flush on his cheeks and an eager intense look in his brilliant eyes. He occupied a semi-recumbent posture and panted violently. The skin was absolutely dry, the temperature was  $99.6^{\circ}$ . The lips were bright coloured, the tongue was very slightly coated by a grayish fur. The patient complained that he did not receive sufficient food to satisfy his hunger. The digestive functions were thoroughly carried out, and the abdominal viscera presented no departure from health. There was no glandular change and the spleen was normal in size. The radial artery was healthy, the vessel was moderately filled, and the blood pressure was low. The rate of the pulse was 135. It was perfectly regular and nearly equal. The carotid arteries pulsated somewhat strongly, and on looking at the præcordia, distinct pulsation could be seen in the third, fourth, and fifth intercostal spaces. On applying the hand, no abnormal impulse could be detected, and there was no thrill. Percussion showed the right border of the heart to be two inches, and the left to be three and a half inches, from the mid-sternal line. The upper border was at the superior edge of the third left costal cartilage. On auscultation in the aortic area, there was a loud yet soft systolic murmur propagated over the whole of the manubrium sterni, towards the upper portion of which it was loudest. This murmur could be heard distinctly in both carotid arteries. In the mitral area a short, sharp, blowing, systolic murmur was heard, which was propagated well into the axilla and towards the angle of the scapula. In the pulmonary area there was slight accentuation of the second sound, and in the tricuspid area a loud, low-pitched, blowing, systolic murmur, propagated up the sternum principally, but in every direction for a short distance; this murmur was quite different in character from that heard either in the aortic or mitral areas.

The patient's breathing varied in rate between 40 and 50 respirations per minute. It was characterised by considerable depth, as well as frequency. Examination of the chest failed to reveal the slightest departure from conditions of health, there being no evidence of hydrothorax or of hyperæmia of the lungs, and it seemed probable that the

dyspnœa was partly produced by erethism, and partly the result of cardiac debility. No other system presented abnormal symptoms, and it may be noted in passing that the patient had been sleeping well at nights.

In this case there could be no doubt that the mitral cusps had undergone some damage on account of subacute endocarditis. The change, however, could not be great, and probably consisted of the presence of a few small vegetations, since there was not the least evidence of aortic incompetence. The mitral and tricuspid valves were obviously incompetent, but the incompetence was probably produced by weakness of the cardiac muscle and not by any organic lesion of the cusps. The worst point about the case was the extreme acceleration of the pulsation and respiration. Tachycardia, as has already been mentioned, is always a symptom of grave prognostic import. In this case, however, both the tachycardia and dyspnœa appeared to Dr. Veitch and myself to be produced more by erethism than by any other cause, and we felt ourselves, therefore, justified in giving a satisfactory prognosis, which was warranted by the result.

CASE 16. *Subacute Endocarditis*.—A. M., aged 20, laundress, was admitted to the Royal Infirmary, 10th July 1893, complaining of pains in her joints.

It was impossible to ascertain whether there were any special family tendencies, since the only facts that could be elicited were, that the patient's father died at the age of 74, after a long illness, and that her mother died, somewhere about the age of 40, quite suddenly. All her brothers and sisters, two and three respectively, were perfectly well. Her social conditions had been satisfactory, but her occupation exposed her to considerable vicissitudes of temperature. The patient had previously suffered three times from subacute rheumatism, and had passed through an attack of pneumonia five or six years before admission. She had, further, had the usual infectious diseases of childhood.

The attack for which the patient came under treatment began three weeks before admission, with pains in the joints. The temperature was 102° on the evening of her entrance into hospital, and for a few days it ranged between 100° and 102°, while the pulse oscillated between 80 and 100. The patient was somewhat pale, and the surface was bedewed with copious perspiration.

The tongue was coated with a heavy white fur. There were, however, no other alimentary symptoms. The radial arteries were healthy; the pulse rather empty, and of moderate tension. It varied in frequency,

as above mentioned, but was always perfectly regular and equal. On inspection of the præcordia, no abnormal symptoms could be detected. The apex beat was situated in the fifth intercostal space,  $4\frac{1}{2}$  in. from mid-sternum. It was unattended by any thrill, and no vibration could be perceived over any part of the præcordia. The cardiac dulness extended  $11\frac{1}{2}$  in. to the right, and 5 in. to the left of the middle line at the level of the fourth costal cartilages. On auscultation, a systolic murmur was heard over almost the whole præcordia. On careful investigation, it was found to resolve itself into several constituents, which, however, blended into each other. There were four points of maximum intensity, one at the apex beat, another at the junction of the fifth costal cartilage with the left edge of the sternum; another at the summit of the manubrium sterni, immediately to the left of the mesial plane; and the fourth occupied the second left intercostal space,  $1\frac{3}{4}$  in. from mid-sternum. The systolic murmur heard over the manubrium was conducted up the carotid arteries.

In this case, it was clear that there was some dilatation along with hypertrophy of the heart, associated with some disturbance of the mechanism of the aortic cusps.

## CHAPTER IX.

### CHRONIC AFFECTIONS OF THE ORIFICES AND VALVES.

By valvular disease is generally understood the entire series of affections of the orifices and valves of the heart. In many of these diseases the valves are only secondarily affected. Very many instances of incompetence, for instance, owe their origin entirely to dilatation of orifices, or, as in the case of the auriculo-ventricular valves, to disturbances of the papillary muscles or tendinous cords; but, inasmuch as inadequacy of the valves is the determining factor in allowing regurgitation, such cases may well be, and usually are, regarded as falling under the group of valvular diseases.

Our knowledge of valvular disease is the result of a gradual growth of information. During the earlier periods of modern medicine, most of the observations connected with valvular disease were concerned almost entirely with morbid anatomy, while during later times pathological investigation and clinical observation have gone hand in hand. Even during the former, there were, nevertheless, some interesting observations. Riverius and Willis in the end of the seventeenth century, and Vieussens, Meckel, and Morgagni during the first half of last century, gave some excellent descriptions of structural changes, along with some clinical details; these are, however, in consequence of their date, necessarily inadequate. In the end of last century, John Hunter, in that monumental work on the Blood to which we owe so much, gave some striking facts in regard to a case of aortic disease.

In the early years of the present century diseases of the



valves were for the first time adequately treated by Corvisart, Burns, and Kreisig. From the standpoint both of morbid anatomy and of clinical teaching, these works arouse admiration. Corvisart's work is remarkable for the excellent account of the morbid appearances in valvular diseases, as well as from his application of the method of physical exploration which he did so much to popularise. Burns not only extended the pathological knowledge of these affections, but, with regard to symptoms, he almost anticipated later discoveries, inasmuch as he describes a hissing sound observed by Brown in valvular disease. It may be said that, if Hooke can be regarded as in some sort a prophet when he looked forward to the possibility of recognising the sounds generated within the human body, so Burns may be held to have vaguely foreshadowed the observations of Laennec. The work of Kreisig, most suggestive as it is in regard to the whole realm of cardiac disease, is perhaps of greatest import in regard to the causation of endocardial disease.

With the appearance in 1819 of the immortal work of Laennec, the new era in the study of diseases of the chest began. Laennec's work, although imperfect and even erroneous in many parts, laid the foundation of all subsequent knowledge, and was followed by the works of Bertin, Hope, and Bouillaud, all of which still further extended the knowledge of valvular disease. These writers have been succeeded by later observers, who will be referred to from time to time in subsequent pages in connection with the special observations for which we owe them our gratitude.

FREQUENCY.—The relative proportion of cases of chronic disease of the orifices and valves to disease in general, as well as the ratio of different forms of disease of the orifices and valves to each other, necessarily varies considerably in different statistics. Lockhart Gillespie has carefully collected and analysed the statistics of the Royal Infirmary of Edinburgh during the last five years, and the results that follow furnish useful information.

During the five years, among 2368 cases of cardiac disease there were 1914 admissions with valvular lesions, of which the following classification has been made:—

## NUMBER OF ADMISSIONS FOR VALVULAR DISEASE.

Lesions.	Aortic Valve.	Mitral Valve.	Two or more Valves.	Tricuspid and Pulmonary.	Total.
Males . . .	354 or 28·5 per cent.	597 or 48·1 per cent.	278 or 22·4 per cent.	12 or 0·96 per cent.	1241
Females . .	57 or 8·4 per cent.	523 or 77·7 per cent.	90 or 13·3 per cent.	3 or 0·5 per cent.	673
Total . . .	411 or 21·47 per cent.	1120 or 58·5 per cent.	368 or 19·2 per cent.	15 or 0·78 per cent.	1914

A glance at the figures will show at once striking differences in the distribution of the male and female cases. In the males, those with mitral lesions only are slightly more than double in number those with lesions of the aortic and mitral valves together, or with these lesions in addition to affections of the tricuspid or pulmonary orifices; while they are rather less than twice as numerous as those with aortic affections only.

The females with purely mitral lesions number 77·7 per cent. of their total, or nearly ten times the number with aortic disease, and six times the number with double lesions.

Taking both sexes together, the cases with purely mitral lesions are more than half the total; those with aortic disease are only slightly more numerous than the cases in which the two or more valves were affected; and the cases of tricuspid or pulmonary valvular disease alone only represent 0·78 per cent. of the total.

The following tables give the analysis of the admissions for valvular diseases, in which, for the sake of economising space, symbols represent the valvular lesions present. These symbols have proved most useful in the ordinary routine work of the Medical Registrar. Incompetence is expressed by a "minus sign," obstruction by a "plus," and a double lesion by a "plus-minus"; the initial letter of the name of the valve is used to designate it; thus A — represents aortic incompetence, M ± mitral obstruction and incompetence, or A ± M —, a case with double aortic lesions along with mitral incompetence.

ADMISSIONS FOR VALVULAR DISEASES.

Age.	AORTIC LESIONS.											
	Males.				Females.				Totals.			
	A -	A +	A ±	Total.	A -	A +	A ±	Total.	A -	A +	A ±	Total.
1-9 .	...	...	...	...	...	...	...	...	...	...	...	...
10-19 .	6	...	7	13	...	...	2	2	6	...	9	15
20-29 .	15	6	22	43	1	3	11	15	16	9	33	58
30-39 .	26	6	34	66	8	1	5	14	34	7	39	80
40-49 .	31	6	58	95	2	1	10	13	33	7	68	108
50-69 .	44	12	70	126	3	5	5	13	47	17	75	139
69+ .	5	...	6	11	...	...	...	...	5	...	6	11
Total .	127	30	197	354	14	10	33	57	141	40	230	411

Age.	MITRAL LESIONS.											
	Males.				Females.				Totals.			
	M -	M +	M ±	Total.	M -	M +	M ±	Total.	M -	M +	M ±	Total.
1-9 .	8	3	6	17	6	2	2	10	14	5	8	27
10-19 .	37	17	24	78	42	44	28	114	79	61	52	192
20-29 .	43	27	22	92	55	65	30	150	98	92	52	242
30-39 .	51	32	23	106	39	31	26	96	90	63	49	202
40-49 .	46	18	17	81	35	16	12	63	81	34	29	144
50-69 .	146	36	28	210	62	13	11	86	208	49	39	296
69+ .	11	...	2	13	4	...	...	4	15	...	2	17
Total .	342	133	122	597	243	171	109	523	585	304	231	1120

## PERCENTAGE OF NUMBER AT AGE TO TOTALS.

AGE.	AORTIC LESIONS.											
	Males.				Females.				Totals.			
	A -	A +	A ±	Total.	A -	A +	A ±	Total.	A -	A +	A ±	Total.
1-9 . .	...	...	...	...	...	...	...	...	...	...	...	...
10-19 . .	4.7	...	3.5	3.6	...	...	6	3.5	4.2	...	3.9	3.6
20-29 . .	11.8	20	11.1	12.1	7.1	30	33.3	26.3	11.3	22.5	14.5	14.1
30-39 . .	20.4	20	17.2	18.6	57.1	10	15.1	24.5	24.1	17.5	16.9	19.4
40-49 . .	24.4	20	29.4	26.8	14.2	10	30.2	22.8	23.4	17.5	29.5	23.8
50-69 . .	34.6	40	35.0	35.5	21.4	50	15.1	22.8	33.3	42.5	32.6	33.5
69+ . .	3.9	...	3.0	3.1	...	...	...	...	3.5	...	2.6	2.6
Per cent. of totals to total for valve	35.8	8.4	55.6	-	24.5	17.5	57.89	-	34.3	9.7	55.9	-

Age.	MITRAL LESIONS.											
	Males.				Females.				Totals.			
	M -	M +	M ±	Total.	M -	M +	M ±	Total.	M -	M +	M ±	Total.
1-9 . .	2.3	2.2	4.9	2.8	2.4	1.1	1.8	1.9	2.3	1.6	3.4	2.4
10-19 . .	10.8	12.7	19.6	13.0	17.2	25.7	25.6	21.7	13.6	20.0	22.5	17.1
20-29 . .	12.5	19.8	18.0	15.4	22.6	38.0	27.5	28.6	16.1	30.2	22.5	21.6
30-39 . .	14.9	24.0	18.8	17.7	16.0	18.1	23.8	18.3	15.3	20.9	21.2	18.0
40-49 . .	13.4	13.2	13.9	13.5	14.4	8.8	11.0	12.0	13.8	11.1	12.5	12.8
50-69 . .	42.6	26.4	22.9	35.5	25.5	7.6	10.0	16.4	37.2	16.1	16.9	26.4
69+ . .	3.2	...	1.6	2.0	1.6	...	...	0.7	2.5	...	0.8	1.5
Per cent. of totals to total for valve	57.2	22.2	20.4	-	46.4	32.7	20.8	-	52.2	27.1	20.6	-



## FREQUENCY.

441

AORTIC AND MITRAL DISEASE.												
AGE.	MALES.								TOTAL.	THREE OR FOUR VALVES AFFECTED.	TRICUSPID.	PULMONARY.
	- K - V	+ K - V	± K - V	- K + V	+ K + V	± K + V	- K ± V	+ K ± V				
1-9 . . .	1	4	1	2	...	1	1	1	4 or 1·4 per cent.	...	...	...
10-19 . .	9	2	...	4	...	11	1	7	37 "	1	...	...
20-29 . .	11	2	...	4	2	19	1	14	54 "	1	5	1
30-39 . .	11	7	...	1	2	9	3	6	39 "	1	2	...
40-49 . .	18	1	1	6	1	1	28	9	67 "	1	1	...
50-69 . .	15	1	2	5	1	35	5	3	67 "	1	3	...
69+ . . .	3	...	...	1	...	...	1	...	5 "	...	...	...
Total . .	68	15	4	19	6	103	15	40	273	5	11	1
FEMALES.												
1-9 . . .	1	1	1	...	...	...	...	...	1 or 1·1 per cent.	...	...	...
10-19 . .	...	3	...	...	...	3	2	8	15 "	...	2	...
20-29 . .	2	6	...	1	...	9	...	5	22 "	...	...	...
30-39 . .	1	1	2	2	2	9	...	...	23 "	...	1	...
40-49 . .	3	...	3	...	...	...	1	2	23 "	...	...	...
50-69 . .	4	1	...	1	2	4	...	2	13 "	1	...	...
69+ . . .	1	...	...	...	...	...	...	...	14 "	...	...	...
Total . .	12	11	5	5	5	25	3	17	89	1	3	...
BOTH SEXES.												
1-9 . . .	2	5	2	...	...	1	1	1	5 or 1·3 per cent.	...	...	...
10-19 . .	9	5	...	2	...	14	2	15	52 "	1	2	...
20-29 . .	13	5	...	5	2	28	3	19	76 "	1	5	...
30-39 . .	12	13	...	2	4	18	3	6	62 "	1	3	...
40-49 . .	21	1	3	8	4	28	3	11	80 "	1	1	...
50-69 . .	19	2	2	6	1	39	5	5	81 "	2	3	...
69+ . . .	4	...	...	1	...	...	1	...	6 "	...	...	...
Total . .	80	26	9	24	11	128	18	57	362	6	14	1

Examination of the figures presented in the foregoing tables shows a marked difference between the incidence of admissions in relation to age, with a remarkable similarity between the percentage admissions at each age of females to males, under the various lesions.

The total admissions for aortic disease begin to rise in a marked manner after the age of 20, rise progressively until 69, and then fall; the admissions of mitral cases rise much more quickly, and reach the maximum between the years of 20 and 29; the figure for the twenty years, 50 to 69, being only slightly greater. The total admissions of other valvular lesions are very similar to the last, with a maximum at 40 to 49 years of age.

In each of these tables the percentage of female admissions at the different ages to the totals exceeds that of the males during the earlier years, in the aortic and combined lesions it is greater up to the thirty-ninth year; in the mitral lesions up to the twenty-ninth. During the later periods, the male numbers predominate.

The figures for the aortic lesions, represented singly, correspond fairly closely as regards the totals for both sexes. Except in aortic obstruction, where the numbers fall from the age of 29 to that of 50, a continuous rise in the percentage of admissions occurs until after the age of 69.

The number of females admitted with aortic incompetence between the years of 30 to 39 reaches 57.1 per cent. of the total females affected by it.

The same excess in the proportions of the female cases over the males is seen in the different mitral lesions up to the thirty-ninth year in mitral incompetence, and double mitral lesions up to the twenty-ninth year in mitral obstruction. In mitral incompetence the admissions are similar to those in aortic lesions. In mitral obstruction, and in cases with both lesions, a greater proportion of both sexes are placed in the earlier decades, with the maximum about 20 to 39. Here again, however, the proportion of male admissions during the later years exceeds that of the females.

Among the cases in which lesions of both aortic and mitral valves were diagnosed, those with aortic incompetence

and obstruction and mitral incompetence were much the most frequent, forming 35 per cent. of the total, 37 per cent. of the male, and 28 per cent. of the female cases. As in the former instances, the males were admitted with these lesions in greater numbers between the ages of 40 and 69, the females chiefly between 20 and 39. Next in frequency are those with incompetence of the aortic and mitral valves, in whom the admissions show a greater proportion of female admissions later in life, of males from 20 to 49 years of age. Double lesions of both valves give the next highest figure, then aortic incompetence and mitral obstruction, and aortic obstruction and mitral incompetence, while the other lesions occur in comparatively small numbers.

Apart from the more infrequent lesions, the greatest proportion of females compared with males is shown in the admissions for aortic incompetence and mitral obstruction, where the female cases form 42 per cent. of the total.

The numbers admitted with lesions of all four valves, or with tricuspid or pulmonary disease, are too few to merit further discussion.

ETIOLOGY.—Chronic affections of the orifices and valves owe their origin to a large series of causes which vary within wide limits in their relative frequency of incidence. Some of these factors undeniably operate in early embryonic conditions, but most of them are connected with acute or chronic processes arising in the course of independent existence.

Many of the influences exerted in the production of valvular diseases are connected with the individuality of the patient, and may be looked upon as predisposing causes; others are imposed upon the system from without, and are, therefore, more or less directly exciting causes. As some of these influences act in a twofold manner, being at once predisposing and exciting, it is not possible to draw any sharp distinction between them.

Due allowance must be made for the influence of heredity in the production of valvular disease. That tendencies to cardiac affections, as originally noticed by Lancisi, are transmitted by parent to child admits of no manner of doubt. It is possible that the older physicians allowed too much weight to such hereditary

transmission, while modern writers have, until recently, attributed too little importance to it. Two observers have in recent years brought forward facts in support of the transmission of a tendency to valvular disease from parent to child. Leube has very clearly stated the intimate connection which rheumatism, chorea, and organic cardiac lesions bear to each other from the standpoint of heredity; while Pierson narrated a series of cases in which he observed the inheritance by children of the parents' predisposition to valvular disease. Many observations of this kind will be found amongst the cases described in other chapters.

Nothing need be said in this connection with regard to the production of valvular disease by means of congenital causes, since these have been treated in a previous chapter. It will, however, be mentioned in the sequel, when reference is made more particularly to some valvular lesions, that it is often a matter of extreme difficulty to determine whether a valvular affection has taken its origin during uterine life, or has been developed at a later period of existence.

Age and sex must be allowed a certain amount of influence in the production of these diseases. Mitral diseases, for example, are more frequently found in early, while aortic diseases are more common in middle life. Aortic disease is more often observed amongst men, and mitral disease amongst women.

Occupations which involve any severe and long-continued muscular stress are apt to lead, both in man and the lower animals, to valvular defects. Not only is stress prone to produce relative insufficiency of the auriculo-ventricular valves by means of cardiac dilatation; it is also apt to lead to chronic endocarditis, as well as to degenerations of the endocardium, more especially of the left side of the heart and of the aorta. Boerhaave long ago observed that wild deer were much more liable to degenerative changes of the endocardium than those reared in captivity, a fact which he attributed solely to the difference in the amount of physical exertion. Men who are engaged in occupations requiring great muscular effort are frequently attacked by aortic disease. The work of Clifford Allbutt on this subject commands unqualified



assent, and in connection with it the remark of Adams may justly be recalled and accepted. In the important paper which he published many years ago, he showed that the reason why the valves of the left side of the heart chiefly suffer is simply on account of their more perfect closure.

Endocarditis is the greatest cause of valvular disease, at least during the earlier periods of life, and it may take its origin, as already seen, in an immense variety of different conditions. Whether acute, subacute, or chronic, it usually leaves behind it some valvular defect, and therefore every factor which has been previously mentioned as leading to endocarditis may produce a chronic valvular affection.

In order to ascertain the relative frequency of the rheumatic origin of organic lesions of the valves, my Birmingham results may be referred to. They deal entirely with instances of undoubted organic disease, and all cases of mitral regurgitation due to relative incompetence of the valves were carefully excluded. The cases were grouped in four classes: (1) cases in which the patient had personally suffered from rheumatism; (2) those in which there was a family history of rheumatism; (3) those with entire absence of any rheumatic history; and (4) those whose history was doubtful. The following summary indicates the results:—

	Mitral.	Aortic.	Both Valves.	Total.
Patient rheumatic . . .	40	8	3	51
Family rheumatic . . .	7	0	0	7
No rheumatism . . .	26	8	0	34
History doubtful . . .	30	6	6	42
	<hr/> 103	<hr/> 22	<hr/> 9	<hr/> 134

Eliminating the 42 cases in which no distinct history can be obtained there remain 92 patients. Adding those cases in which the patient was rheumatic to those in which there was a family history of rheumatism, there are 58 rheumatic as against 34 non-rheumatic cases. Of the total number of patients, therefore, with a distinct history, 63·05 per cent. were rheumatic and 36·95 per cent. were not. Of the cases of mitral disease, 47 were rheumatic and 26 non-rheumatic. Aortic disease was associated with rheumatism in 8 cases, and in 8 there was no rheumatic history.

It is possible that hæmorrhage may sometimes be the starting-point of valvular lesions. The aortic and pulmonary valves can scarcely from the nature of their structure be affected in this way, but the auriculo-ventricular valves, possessing blood vessels, are sometimes the seat of hæmorrhage. The earliest observation of this kind, so far as is known to me, was made by myself, but it was followed shortly afterwards by the publication of a case of great interest by Garel. What relation exists between the blood vessels and valvular disease, it is at present impossible to say, and whether valvular hæmorrhage may be the starting-point of other affections cannot as yet be determined.

The factors which are operative in the production of degenerative changes in the endocardium are among the common causes of valvular disease. Many degenerative changes ensue simply as part of the retrograde tendencies of advancing years, and are more common in certain families which evince a proclivity towards such changes. Long-continued physical stress has been already referred to and need not occupy further time in this connection.

The presence of toxic chemical substances in the blood, whether introduced from without, as in the case of alcohol, lead, and other poisons, or taking origin within the system, as in the case of the arthritic diathesis or renal disease, are powerful agents of this kind. Some infectious diseases have also like effects; the only one of these, however, frequently met with is syphilis.

Valvular derangements, in which the cusps themselves may be perfectly healthy, may have their origin in causes acting entirely upon the orifices, the walls, or the papillary muscles. Severe stress of the heart from physical exertion may give rise to sudden or gradual strain of the cardiac structures. Both acute and chronic disease may produce weakness and loss of elasticity of the orifices, walls, or papillary muscles. Structural alterations of the myocardium, whether myocarditic or degenerative, allow stretching of the textures. Such are types of the factors which may give rise to relative incompetence of the cusps, as will be more fully discussed in a subsequent chapter.

The fact must not be overlooked that many causes operate in more than one way, and thus the study of the etiology of valvular disease becomes somewhat complicated.

Valvular diseases take their origin occasionally in direct or indirect violence. The earliest trustworthy observations on this subject are those of Corvisart, who was followed by Laennec, Bertin, and Bouillaud. The most important of recent writers on this subject are Foster and Barié. The interesting clinical observations of the former will be mentioned in the chapter on aortic disease. The latter in an extensive and comprehensive memoir gave a complete account of all the published instances of valvular rupture, to which he added seven previously unrecorded cases.

Barié divided the injuries into two classes: spontaneous, caused by effort on the part of the patient, and traumatic, in which the injury arose from external agency. The total number of cases collected by him is 38. Of these 19 affected the aortic valves, 10 of which were spontaneous, and 9 traumatic. In 11 of the aortic cases autopsies were obtained, and it was found that 6 had rupture of two valve segments, while 3 had but one segment injured. The segment most frequently damaged was the right, *i.e.* that next the interventricular septum. The mitral apparatus suffered in 16 cases, of which 13 were spontaneous, and 3 traumatic. Autopsies were performed in 15 of these instances; 9 showed rupture of the tendinous attachments of the valves, 5 laceration of the muscular columns, and 1 injury both to chordæ and columnæ. In no case was there any damage to the valves themselves. There were 3 cases of injury to the tricuspid apparatus, 2 spontaneous, and 1 traumatic. In these cases, once a valve was damaged, once the fleshy columns were torn, and once also the tendinous attachments gave way. In the whole series it is quite evident that spontaneous rupture is more frequent than traumatic, their numerical relationship to each other being 25:14. The causation of spontaneous ruptures was found in great muscular efforts at work, the strain of a bad cough, or frantic attempts at respiration when suffocation was imminent. The traumatic ruptures were partly due to direct, and partly to indirect violence.

Barié has given considerable attention to the pathogenesis of these ruptures, and thinks that the surest key to their solution lies in a study of the physiological mechanism of violent efforts. Right-sided rupture is brought about in this way: every effort raises the pressure in the pulmonary circulation, and therefore increases that of the right cardiac cavities. If any violent stress is produced, such as coughing, during ventricular systole, the tricuspid valve may give way. To me it seems that there must be some tricuspid peculiarity before rupture could occur, as the usual result is free regurgitation when the pulmonary pressure rises; it is, however, quite credible that the tension of the whole thorax in certain rare instances may keep the heart from any excentric yielding. The valves of the left side are believed by Barié to undergo rupture by means of augmented pressure, due partly to peripheral resistance, and partly to increased tension on the thoracic and abdominal aorta during great effort.

He thinks that traumatic rupture of the tricuspid and mitral apparatus occurs during the diastolic period, just when the ventricles are nearly or quite full and before the sigmoid valves can open to the sudden and great increase of pressure. In the same way he considers that rupture of the aortic segments must be caused during diastole also, when the aorta is fully distended and no escape can be found save by an abnormal channel.

Experimentally Barié found that it was impossible to cause rupture of the pulmonary valves, and the same may be said of the tricuspid; for the only instance of injury which he was able to produce occurred in the body of a woman, aged seventy-nine, who died in the last stage of cancerous cachexia. The mitral valve was difficult to rupture; the easiest of all being the aortic. In this last it is interesting to note that the segment which most frequently yielded was the right, or that of the interventricular septum.

**MORBID ANATOMY.**—The appearances in chronic affections of the valves form a group whose extremes are widely separated, but which are nevertheless bound together into an almost perfect series by connecting links. Some of the lesions are primarily those of the valves themselves, some are



only connected with the valves in a secondary manner. It will be convenient to classify the structural changes according as they result from endocarditis, degeneration, neoplasms, or traumatism.

*Endocarditic Changes.*—Obstruction may be produced by a thickening of the membrane at the orifice itself, or of that of its cusps. This thickening is usually accompanied by a very considerable growth of fibrous tissue, and there is a tendency to calcification of the affected region. In the case of the cuspid valves the chordæ tendineæ frequently present thickenings from fibrous or calcareous changes. Such changes are shown in one of the illustrations of mitral obstruction, as may be seen in Fig. 147, on page 523.

Adhesions between the cusps are of frequent occurrence, and lead, more particularly in the case of the auriculo-ventricular valves, to so much union as to leave extremely restricted orifices, often called, from their appearance, “button-hole” orifices. These adhesions begin at the angle where the neighbouring cusps are in contact, and from that point the union spreads along the edge of the cusps to a greater or less extent. Although more common with regard to the mitral valves than any other, it is not uncommon to find the same condition in the aortic cusps, as seen in Fig. 138, on page 475; in the congenital affections of the pulmonary orifice and its valves a similar lesion is often seen, as has been beautifully figured by Peacock.

Vegetations situated either at the orifice or upon the cusps are extremely frequent causes of obstruction. These, as has already been stated with regard to endocarditis, are found upon the surface which is at once exposed to the current of the blood and to contact with an opposed cusp; they form festoons, as it were, a short distance from the edge of the cusps along the line of apposition. Sometimes those granulations are of small size, and are attended by a considerable amount of shrivelling and shrinking. At other times they are extremely large, and form prominent masses projecting from the surface. A good example of such vegetations is shown in Fig. 137, on page 474.

Aneurysmal dilatations not infrequently occur in the sub-

stance of the valves; but this is far less common in chronic than in fatal acute cases of endocarditis. The mechanism by which such aneurysms are produced is apparently through the formation of ulcers, upon which the blood pressure acts in such a way as to produce pouches. This process is more



FIG. 136.—Hæmorrhage into the tricuspid valve of a sheep,  $\times 40$ . The section shows proliferation of the endothelial cells, and exudation of leucocytes into the clot, with commencing organisation.

commonly found in connection with the mitral valve than in the case of any other.

*Hæmorrhagic Processes.*—As in some degree connected with vascular processes, a word may be said on valvular hæmorrhages, such as have been described by myself and Garel. The only instance which has come under my notice was a small purple mass, about the size of a pea, upon the auricular surface of one of the right auriculo-ventricular cusps of a sheep. The endocardium covering it was perfectly healthy. The microscopic appearances, which are shown in the accom-

panying illustration (Fig. 136), are those of a blood-clot undergoing organisation. Garel's observation concerned the tricuspid valve of a man, on which was a mushroom-like mass, consisting of a large extravasation associated with some vegetations.

*Degenerative Lesions.*—The results of degenerative processes so frequently constitute the lesions in chronic valvular disease that it is impossible to agree with the dictum of Letulle:—"Dans le langage courant, affection valvulaire est synonyme d'endocardite valvulaire chronique." The most common form of this kind of change is sclerotic thickening of the membrane. Patches of thickening, often with some elevations upon the surface, are to be seen. They consist of masses of firm tissue, often indistinctly organised or even almost homogeneous, and are to be regarded as analogous to the lesions of arterio-sclerosis. It is possible that they are produced by reaction processes, to compensate for loss of normal structural characters. The sclerotic patches are often associated with fatty or calcareous changes, and are then commonly termed atheroma.

Fatty changes are not infrequent, more particularly in cases of fatal anæmia and some forms of marasmus. Such alterations are more common on the endocardium of the left side, and more especially of the mitral cusps. They form larger or smaller patches upon the opposed surfaces of the cusps, and give rise to valvular disturbances. Other degenerative processes are occasionally found, but are relatively so uncommon as to merit no attention in this connection.

*Neoplasms.*—New formations are rare sources of obstruction. Endocardial tumours, as will be afterwards mentioned, are not by any means uncommon, but those which interfere with the valvular apparatus are certainly rare. Sometimes such tumours have their origin in the walls of the heart, as in the well-known case recorded by Gairdner, in which tricuspid obstruction was diagnosed ten years before the death of the patient, or they may arise from the valves themselves. Garel has collected the observations which have been made upon this subject in the paper already referred to.

*Traumatic Lesions.*—The changes produced by injury have been already fully mentioned as regards the relative propor-

tion of different lesions. The characters of the lesions found after death depend upon the situation of the injury—whether it affects a cusp or a tendinous cord—and upon the length of time which has elapsed since the violence. In most instances the primary lesion has been followed by the development of vegetations, and the result has been the production of obstruction and incompetence.

Incompetence may be directly produced by affections of the cusps themselves such as have just been described. As the result of both acute and chronic processes there may be a loss of substance from ulcerative processes, or the cusps may become shrivelled and shrunken, or they may be bound by adhesions in such a way that they fail to close the apertures correctly.

Disturbances of the functions of the valves can be produced by causes other than those directly affecting the cusps. The orifices may be widened so that the cusps will not meet, or, as happens in the case of the auriculo-ventricular valves, the papillary muscles and the tendinous cords may be in fault, or the ventricles may be so dilated as to produce a want of correspondence in the relation of the cavities and the valvular arrangements. Stretching of the orifices may have its origin in simple strain from stress—the so-called overstrain of the heart—or in loss of elasticity resulting from weakness, or in acute or chronic structural alterations of the myocardium. Atrophic and degenerative processes affecting the papillary muscles diminish their different performances of function, while the tendinous cords may stretch or shrink in consequence of disease. Dilatation of the cavity of the heart may produce a want of proper adjustment of the valvular mechanism, and, as a result, regurgitation is allowed at the auriculo-ventricular orifices.

All these are examples of what is often termed functional or relative incompetence of the valves, in which the cusps themselves may be perfectly healthy. All of them, as will be seen in a subsequent part of this work, are more common on the right side of the heart than on the left, and they are practically unknown in connection with the aortic orifice.

**EFFECTS.**—Many secondary results follow affections of the



orifices and valves, some of which are more or less directly connected with the heart itself, while others are remote consequences produced by disturbances of the circulation. In that part of this work dealing with the general effects of diseased processes upon the circulation, the chief considerations have been fully dealt with, and some of the effects will be again discussed in those chapters which are devoted to the special lesions of the valves. It is, therefore, unnecessary in this place to do more than refer briefly to some points which require slight amplification.

The chronic processes affecting the orifices and valves have by no means the same tendencies to spread by extension as is the case in regard to the acute and subacute forms of endocarditis, and the effects which are produced upon the heart itself are brought about by more indirect processes. There are, nevertheless, some direct effects upon the heart, as for instance the morbid changes set up by friction which were pointed out by Hodgkin. In the case of large indurated masses floating with a certain degree of freedom in the blood current, it is no uncommon thing to find that, at the points of impact, where they come in contact with the interior of the heart or great vessels, there are thickenings and roughenings of the surface.

It may be held as a general principle that there are some characteristic differences between the effects of obstruction and incompetence. Such is the case in principle, but, as has been shown previously, the results of Hamilton's careful investigations show some effects which would not altogether be expected.

Beyond the obstructive orifice there must be less blood supply, and in consequence there will be diminished blood pressure, and a tendency to lowering of the general nutritive processes. Behind the obstructed orifice there is a considerable amount of hypertrophy, without any great degree of dilatation.

Beyond valves which are incompetent there is not necessarily any reduction in the amount of blood sent out into the general circulation, but in consequence of regurgitation there is a diminution of the mean blood pressure, which more particularly affects the peripheral circulation. Behind the

incompetent valves there is dilatation with hypertrophy. The dilatation is much greater than behind pure obstruction.

Certain structural alterations of the cardiac walls, chiefly chronic myocarditis and myocardial degeneration, are apt to ensue in consequence of long-continued disturbance of the valvular mechanism. The full discussion of dilatation and hypertrophy must be reserved until the affections of the myocardium are taken up, and the same may be said in regard to the degenerations of cardiac failure.

Diseases of the orifices and valves of the left side of the heart lead to disturbances of the pulmonary circulation, with dilatation of the veins and capillaries of the lung, resulting in venous hyperæmia and œdema, which are in turn followed by dilatation of the pulmonary artery and its branches, frequently leading to atheromatous changes in their walls.

Affections of the orifices and valves of the right side of the heart give rise to dilatation of the venæ cavæ and their tributaries, leading to engorgement of the neck, legs, and internal viscera, as shown by cyanotic changes in the solid organs, and catarrh of the mucous membranes.

In consequence of the reduction of the rapidity of the circulation, the blood is apt to coagulate both in the chambers of the heart, and in the great venous channels, so that clots are produced, which may be coloured, or colourless, according to the date of coagulation.

Embotic processes may arise not only as regards coagula formed within the heart, but also in respect of vegetations affecting the valves. Hæmorrhage may result from any of the engorged blood vessels, but it is much more frequently found in the lungs than in any other situation.

**SYMPTOMS.**—It may be stated as a broad general principle that, with the exception of the physical signs caused by diseases of the valves and orifices, these lesions do not of themselves directly produce characteristic symptoms; it is only when their secondary effects are produced that they give rise to any characteristic clinical features. These effects have already been fully analysed, and require but little consideration in this place. Their association with the special valvular lesions will be discussed in subsequent chapters.

**DIAGNOSIS.**—The differentiation of the various valvular lesions, whether single or combined, must be considered when treating of the different diseases.

**PROGNOSIS.**—The outlook in chronic valvular disease can only be correctly estimated after a thorough consideration of every factor which has influence upon the present, and may affect the future condition. In making inquiry into the conditions and relations of any given case of valvular disease every influence must be carefully investigated.

The family history of the patient is of real influence, since from the family tendencies a prophetic light may be cast upon the prospects of the patient. A family proclivity to long life or to early death is of much service in forming an estimate of the outlook, and more particularly so when parents or ancestors have been victims of valvular disease.

Age is a factor of great importance, since valvular diseases, which in themselves during the active period of life may not be serious, assume a much graver aspect at the extremes of life; more especially is this to be considered in dealing with valvular disease in childhood. It is no uncommon experience to meet with instances of mitral obstruction during childhood, and the fact cannot be gainsaid that in a very considerable proportion of such patients a fatal termination occurs during the readjustment of all the bodily functions at the time of puberty or adolescence.

The occupation, habits, and surroundings of patients suffering from valvular disease are points which must be taken into consideration. It need only be said in this place that if these are satisfactory the prognosis will be by so much the better, and the converse also holds good.

The medical history of the past will in every case furnish indications of utility. This not only gives information as to the power of resisting the disease evinced by the individual—a consideration of much importance—but the presence or absence of certain conditions must be ascertained.

The abuse of alcohol, a tendency to the uric acid diathesis, the presence of any renal affection or of specific affection—these and many other analogous considerations must be taken into account.

The nature of the lesion, its mode of origin, the length of its duration, and its effects upon the heart itself and the system at large, are most important matters directly connected with valvular disease.

Great differences exist in the gravity of different lesions. Aortic obstruction, for instance, is much less serious than mitral obstruction, while aortic incompetence is very much graver than mitral incompetence.

The extent of the lesion is one of the most difficult matters to determine. Whether a serious obstruction or a free regurgitation is present is, as will be seen in the sequel, difficult indeed to determine. The evidences furnished by physical examination of the heart itself do not, as a general rule, throw any light upon this subject, and the nearest approach to exact information is to be derived from a study of the effects of the lesion upon the heart and circulation.

Referring to the Edinburgh statistics of Lockhart Gillespie, it will be seen that the incidence of mortality with regard to age differs markedly from the incidence of admissions.

In the tables for all the cases with aortic lesions, no deaths are shown until after the age of 19. The rates for each period until 69 is passed are very similar; the male results, however, exhibit a marked rise between the years of 20 and 29; and the females show a great excess over the males from the age of 39 to 69. The maximum mortality among males falls between 20 and 29, among females between 40 and 49. The maximum mortality in both sexes taken together, from aortic incompetence, occurs between 50 and 69; from aortic obstruction, between 40 and 49, as also in double aortic lesions. The greatest proportion of males with aortic incompetence or obstruction occurs between 50 and 69, but in those with double lesions the years from 20 to 29 are the most fatal. The female maximum in aortic incompetence and aortic obstruction falls between the years 49 and 50, and in the first is much higher than in the males; the 100 per cent. at that age in aortic obstruction is only fortuitous, the one female case admitted at that age having died. In double aortic lesions no deaths among females are recorded, except between the ages of 40 and 69.



The figures representing the mortality from all mitral lesions in both sexes rise progressively with the age, the female rate always a little over that of the males, until after the age of 69, when three deaths out of four female admissions raise it to 75 per cent. The rates in mitral incompetence are very similar to those for all mitral cases, except for a more pronounced mortality between 30 and 39. Mitral obstruction proves most fatal from 30 to 39 in the males, and from 40 to 49 in the females. The female rate between 20 and 29, 40 and 49, and 50 to 69 is higher than in males. In cases of double mitral lesions both males and females show a death-rate between the years 10 to 19 above that for the next decade. The male maximum falls between 30 and 49, the female between 50 and 69. The highest rate for the two sexes occurs between 50 and 69.

Aortic incompetence with mitral lesions proved most fatal between 1 and 9 years, where one case out of two admissions died. The decade between 30 and 39 shows the high rate of 44·4 per cent., due to a rate of 50 per cent. in the males, and 33·3 per cent. in the females. In the females, however, the rate is higher between 40 and 49—40 per cent. Where aortic obstruction is associated with mitral lesions, few deaths fall to be recorded, and all of these between the years 20 and 49. The highest rate here is 33·3 per cent. in males between 30 and 39. The death-rates in cases of double aortic with mitral lesions are highest—30·9 per cent.—between 40 and 49; lowest—9·6 per cent.—between 10 and 19; while no deaths are recorded between 1 and 9 of the three cases admitted, or in the case of the one admission over 69 years of age.

Five of the six cases reported as suffering from lesions of more than these two valves proved fatal, or 83·3 per cent.; while of the fourteen admissions in which symptoms of tricuspid incompetence were found, three died—all of them males—giving a death-rate of 21·4 per cent. of the total, or 27·2 per cent. of the males.

In order to ascertain what amount of correspondence obtains between the current conceptions regarding the gravity of different lesions and the facts observed in the Royal Infirmary, the tables of Lockhart Gillespie may be laid

under contribution. The following table gives the mortality of different valvular lesions:—

NUMBER OF DEATHS AND MORTALITY AMONG THE CASES  
OF VALVULAR DISEASE.

Lesions.	Aortic Valve.	Mitral Valve.	Two or more Valves.	Tricuspid and Pulmonary.	Total.
Males . . .	52 or 14·6 per cent.	72 or 12 per cent.	67 or 24·1 per cent.	3 or 25 per cent.	194
Females . .	10 or 17·5 per cent.	69 or 13·2 per cent.	20 or 22·2 per cent.	...	99
Total . . .	62 or 15·0 per cent.	141 or 12·5 per cent.	87 or 23·6 per cent.	3 or 20 per cent.	293

The highest mortality occurs among the cases with both aortic and mitral disease, and is rather higher among the males. The aortic cases present a higher rate than the mitral, while here the disparity between the rates among the males and the females is greatest, being in fact in the females 2·9 per cent. over the figure for the deaths among males, or 20 per cent. of an excess compared with the male rate.

The fatal cases in relation to age and lesion are embodied in the following table:—

DEATHS FROM VALVULAR DISEASES.

MALES.															
Ages.	A -	A +	A ±	All A.	M -	M +	M ±	All M.	A - M -	A - M +	A + M -	A + M +	A ± M -	A ± M +	A ± M ±
1-9 . . .	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1
10-19 . . .	..	..	..	..	1	..	2	3	1	2	..	..	1	1	6
20-29 . . .	3	..	5	8	4	2	..	6	2	..	..	1	4	..	11
30-39 . . .	3	..	6	9	7	5	5	17	6	3	1	..	2	..	14
40-49 . . .	4	..	10	14	5	2	4	11	3	..	..	1	8	1	15
50-69 . . .	10	2	8	20	25	2	5	32	4	..	..	..	10	1	16
+69 . . .	1	..	..	1	3	..	..	3	..	..	..	..	..	..	..
Total .	21	2	29	53	45	11	16	72	17	5	1	2	25	3	63

FEMALES.															
Ages.	A -	A +	A ±	All A.	M -	M +	M ±	All M.	A - M -	A - M +	A + M -	A + M +	A ± M -	A ± M +	A ± M ±
1-9 . . .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
10-19 . . .	..	..	..	..	4	..	3	7	..	..	..	..	..	..	..
20-29 . . .	..	..	..	..	4	7	2	13	..	1	..	..	1	1	2
30-39 . . .	3	..	..	3	9	3	3	15	..	3	..	..	1	3	7
40-49 . . .	1	1	2	4	4	4	2	10	1	..	1	1	..	1	5
50-69 . . .	1	1	1	3	16	1	4	21	..	..	..	..	..	..	1
+69 . . .	..	..	..	..	3	..	..	3	1	..	..	..	..	..	1
Total .	5	2	3	10	40	15	14	69	2	4	1	1	1	4	19

TOTALS.															
Ages.	A -	A +	A ±	All A.	M -	M +	M ±	All M.	A - M -	A - M +	A + M -	A + M +	A ± M -	A ± M +	A ± M ±
1-9 . . .	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1
10-19 . . .	..	..	..	..	5	..	5	10	1	2	..	..	1	1	6
20-29 . . .	3	..	5	8	8	9	2	19	2	1	..	1	5	1	16
30-39 . . .	6	..	6	12	16	8	8	32	6	6	1	..	5	..	21
40-49 . . .	5	1	12	18	9	6	6	21	4	..	1	2	8	2	20
50-69 . . .	11	3	9	23	41	3	9	53	4	..	..	..	10	1	17
+69 . . .	1	..	..	1	6	..	..	6	1	..	..	..	..	..	1
Total .	26	4	32	62	85	26	30	141	19	9	1	3	29	5	82

Useful information may be derived from the consideration of the relation between the number of deaths to the number of admissions which is embodied in the following table:—

PERCENTAGE MORTALITY IN VALVULAR DISEASES TO  
ADMISSIONS AT AGE.

BOTH SEXES.														
Age.	AORTIC.				MITRAL.				AORTIC AND MITRAL WITH					
	A -	A +	A ±	All A.	M -	M +	M ±	All M.	$\frac{M-}{M+} \pm \frac{M\pm}{M+}$ A -	$\frac{M-}{M+} \pm \frac{M\pm}{M+}$ A +	$\frac{M-}{M+} \pm \frac{M\pm}{M+}$ A ±	All A and M.	Three Valves.	T -
1-9 .	..	..	..	..	..	..	..	..	50	..	..	20	..	..
10-19 .	..	..	..	..	6·3	..	9·6	5·2	18·7	..	9·6	11·5	100	..
20-29 .	18·7	..	15·1	13·7	8·1	9·7	3·8	7·9	16·6	12·5	24·0	21·0	100	..
30-39 .	17·8	..	15·3	15	17·7	12·6	16·2	15·8	44·4	25·0	25·9	33·8	..	..
40-49 .	15·1	14·2	17·6	16·6	11·1	11·3	10·9	14·6	20·0	15·3	30·9	25	100	100
50-69 .	23·4	11·7	12	16·5	19·7	6·1	23	17·9	17·3	..	26·5	20·9	100	66·6
over 69	20	..	..	9·1	60	..	..	35·2	25·0	..	..	16·6	..	..
Total .	18·4	10	13·9	15	14·5	8·2	13	12·5	25·2	11·3	23·1	22·6	83·3	21·4
MALES.														
1-9 .	..	..	..	..	..	..	..	..	100	..	..	25	..	..
10-19 .	..	..	..	..	2·7	..	8·3	3·8	21·4	..	15	16·2	100	..
20-29 .	20	..	22·7	18·5	9·3	7·4	..	6·5	15·4	14·2	23·5	20·3	100	..
30-39 .	11·5	..	17	13·6	13·3	15·6	21·7	16	50	33·3	22·2	35·9	..	..
40-49 .	12·5	..	17·2	14·7	10·8	11·1	23·5	13·5	15	12·5	28·1	22·3	100	100
50-69 .	22·7	16·6	11·4	15·9	17·5	5·5	17·9	15·2	22·2	..	27·9	23·1	100	66·6
over 69	20	..	..	9·09	27·2	..	..	23	..	..	..	..	..	..
Total .	16·5	6·6	13·7	14·6	13·1	8·2	13·1	12	25·2	10·7	24	23	80	27·2
FEMALES.														
1-9 .	..	..	..	..	..	..	..	..	..	..	..	..	..	..
10-19 .	..	..	..	..	9·5	..	10·7	6·1	..	..	..	..	..	..
20-29 .	..	..	..	..	7·3	10·7	6·6	8·6	20	..	25	22·7	..	..
30-39 .	37·5	..	..	29·4	2·3	9·6	11·5	15·6	33·3	20	33·3	30·4	..	..
40-49 .	50	100	20	30·9	11·4	25	16·6	15·9	40	20	66·6	88·4	..	..
50-69 .	33·3	20	20	23	25·4	7·6	36·3	24·4	..	..	16·6	7·1	100	..
over 69	..	..	..	..	7·0	..	..	75·0	100	..	..	100	..	..
Total .	35·7	20	9	17·5	16·4	8·9	12·8	13·2	25	12·5	22·2	21·3	100	..

Taking the general facts first, aortic lesions are more fatal than mitral, but double lesions show a much greater



mortality than either. In males, aortic lesions do not show nearly such a high death-rate as in females, while that of mitral cases is also less. Lesions of both valves prove more fatal in males than females. Of aortic lesions, aortic incompetence shows the highest death-rate, chiefly due to the great mortality among females—35·7 per cent., against 16·5 per cent. in the males. Aortic obstruction again causes 20 per cent. of deaths among females, but only 6·6 per cent. among males; while double aortic lesions prove fatal in 13·7 of every hundred males, and in 9 in each hundred females. Similarly, mitral incompetence and mitral obstruction give a higher mortality among females; double mitral lesions among males; in the total of both sexes, mitral incompetence proves most fatal, with double lesions close behind it. In cases of aortic and mitral disease, those with aortic incompetence and each of the three mitral lesions prove most fatal—25·2 per cent.—and equally so in both sexes; next in order, cases with aortic obstruction and regurgitation in addition to mitral disease, and here the death-rate among the males exceeds that of the females; where there is aortic obstruction along with mitral lesions the rate is lower, but that among the females exceeds that among the males.

The rate of mortality for double lesions in each individual instance is as follows:—

MORTALITY PER CENTUM.

		A - M -	A - M +	A - M ±	A + M -	A + M +	A + M ±	A ± M -	A ± M +	A ± M ±	Total.
DEATH-RATE.	Male . . .	25·0	33·3	...	5·2	33·3	...	24·2	20·0	25·0	23·0
	Female . . .	16·6	36·3	20·0	...	20	16·6	16·0	66·6	23·5	21·3
	Total .	23·7	34·6	11·1	4·1	27·2	11·1	22·6	27·7	24·5	22·6

The cases in which aortic incompetence and mitral obstruction were diagnosed show the highest mortality in the total for both sexes, and for each sex singly; next, those suffering from a double aortic lesion and mitral obstruction,

due to two out of the total three female cases; those with obstruction of both valves show a very similar mortality; while double lesions of both valves, incompetence of both valves, and double aortic lesions with mitral incompetence, follow in that order and close together. Only one patient died, of those diagnosed as having aortic obstruction and mitral incompetence.

#### THE DEGREE OF MORTALITY FROM THE VARIOUS LESIONS.

	Male.		Female.		Total.	
1	A - M +	33·3 per cent.	A ± M +	66·6 per cent.	A - M +	34·6 per cent.
2	A + M +	33·3 "	A - M +	36·3 "	A ± M +	27·7 "
3	A - M -	25·0 "	A -	35·7 "	A + M +	27·2 "
4	A ± M ±	25·0 "	A ± M ±	23·5 "	A ± M ±	24·5 "
5	A ± M -	24·2 "	All A & M	21·3 "	A - M -	23·7 "
6	All A & M	23·0 "	A +	20·0 "	All A & M	22·6 "
7	A ± M +	20·0 "	A - M +	20·0 "	A ± M -	22·6 "
8	A -	16·5 "	A + M +	20·0 "	A -	18·4 "
9	All A	14·6 "	All A	17·5 "	All A	15·0 "
10	A ±	13·7 "	A - M -	16·6 "	M -	14·5 "
11	M -	13·1 "	A + M ±	16·6 "	A ±	13·9 "
12	M ±	13·1 "	M -	16·4 "	M ±	13·0 "
13	All M	12·0 "	A ± M -	16·0 "	All M	12·5 "
14	M +	8·2 "	All M	13·0 "	A - M ±	11·1 "
15	A +	6·6 "	M ±	12·8 "	A + M ±	11·1 "
16	A + M -	5·2 "	A ±	9·0 "	A +	10·0 "
17	A - M ±	...	M +	8·9 "	M +	8·2 "
18	A + M ±	...	A + M -	...	A + M -	4·1 "

The mode of origin of any valvular lesion is somewhat helpful, since as a rule those which arise in consequence of slow sclerotic processes are of less serious consequence for a considerable length of time than those which arise from endocarditic processes. One reservation, however, must be made, since degenerative lesions of the aortic cusps are somewhat likely to cause coronary changes of evil import.

Endocarditic processes are always liable to be lighted up once more, and to bring about disastrous results.

From the length of time which has elapsed since the commencement of any valvular disease, it is possible to predicate to some extent what will be the probable future of the lesions; for, in cases which have existed for a considerable

time without serious inconvenience, there will naturally be a hopeful expectation. The observations of Clark on this subject are of great value.

The effects of any valvular lesion upon the heart itself further require attention. When a tendency to dilatation disturbs the compensatory process of hypertrophy, the prognosis is serious, and any tendency to cardiac failure or asystole is of evil omen. The condition of the apex beat, the possibility of producing distinct cardiac sounds or loud murmurs, are points to which particular attention will be given in the sequel.

The effects of the lesion upon the system elsewhere must be judged by the condition of the digestion, of the blood, of the lungs, of the kidneys, and of the brain. Symptoms of disturbances of the functions of these organs, such as will be fully detailed below, render valuable evidence as to prognosis.

TREATMENT.—It is scarcely necessary to make the statement that a valvular lesion in itself does not call for treatment. The fact has already been sufficiently insisted on that experimental lesions of the cusps do not produce any alteration in the blood pressure of healthy animals, and this alone is enough to show that affections of the orifices and valves do not, as such, require treatment. It is only when they are associated with other lesions, or are followed by consecutive disturbances, that it is necessary to interfere.

From the nature of the affections now under consideration, it must be obvious that prophylaxis, in the correct sense of the term, is scarcely applicable to the matter in hand. The subject under discussion is that of chronic lesions, most of which rarely come under observation until fully developed. It occasionally happens, no doubt, that patients are seen, in whom there is some evidence of the progress of a valvular lesion undergoing insidious development, and in whose case, therefore, questions of prevention may arise. This is, however, at most, one of the less common experiences in cardiac disease. The case of chronic valvular disease is altogether different from that of endocarditis, which has already been fully discussed from every point of view.

If prophylaxis, in the strict acceptation of the word, is not a matter for consideration at present, there are, notwithstanding, some questions as to the management of valvular affections closely related thereto. It must be clear that with any damage to the valvular mechanism—no matter how healthy may be the heart wall on the one hand, the vessel wall on the other—the entire apparatus is working at a disadvantage as compared with one in which the whole structures are normal, and the very processes by which the disturbances are compensated lead inevitably in the long-run to disaster. The circulation, in short, labours under increased stress, which is apt to lead to strain. In any case, therefore, it is necessary to obviate every baneful influence which may operate upon the circulation, and to avert, in so far as may be possible, the injurious effects of any lesion which may be present.

When attempting to make a forecast of the future, and to lay down rules for the guidance of any case of valvular disease, the age and sex of the patient will necessarily set natural limits to the possibilities of management; it is clear that the conditions are subject to entirely different agencies when a valvular disease is found in early, middle, or later life, and in almost the same way the sex of the patient exercises a preponderating influence. In a similar manner, the environment and occupation will require the most careful consideration in regard to their effects upon the individual. In every case, further, the family history and previous health of the patient will be found full of importance, as showing the tendencies which have to be dealt with.

Cases of cardiac disease naturally fall into two classes—those in which compensation for valvular lesions is adequate, and those in which it is insufficient.

A few words may be said as regards the general treatment of patients with adequate compensation. In such cases a certain degree of watchful care must undoubtedly be exercised, while *nimia diligentia* must be the motto. Every factor which can exert influence upon the future life of such patients must be carefully taken into consideration. The residence may have to be selected with a view towards the avoidance of extremes of temperature or vicissitudes of climate. The diet



should certainly be regulated so as to prevent the ingestion of too much nitrogenous food, while the quantity of alcohol should be strictly arranged. The occupation should, if possible, be one which will allow of sufficient open-air life to keep up the nutritive processes. The amount of exercise and rest must be fixed well within healthy limits, while the general environment should be, in so far as may be possible, cheerful and salubrious.

Young patients are not infrequently brought before the physician in order to seek advice upon all the foregoing points. The question which will probably in all such cases weigh most heavily is that of the future occupation of the patient. On receiving information of the existence of some valvular defect, the relatives are frequently tempted, by a policy of despair, to withdraw the patient from the world altogether as a valetudinarian. Such a tendency as this, however natural it may be, should be strenuously opposed. Many valvular lesions are not incompatible with the performance of duties involving a considerable amount of exertion, and much even of the best work which the world has seen has been contributed by those who have suffered under valvular disabilities. In this connection, the results and opinions of Stokes and Clark may be referred to as emphasising views resembling those to which expression is now given.

In the other class of cases, when compensation is not adequate, either from a primary weakness of the myocardium or a secondary failure in consequence of long-continued stress, masterly inactivity is no longer to be thought of. When the various symptoms of cardiac failure are present, they must be combated with the most sedulous attention by every means within reach of the physician. There are many different ways in which the failure of compensation may show itself, and the groups of symptoms thereby produced have been already sufficiently analysed. The one principle upon which attention must now be concentrated is that, no matter whatsoever such symptoms may be, they are one and all associated with, and mostly dependent upon, the result of diminished arterial pressure. One general scheme of treatment is therefore applicable to almost all conditions arising from valvular

disturbance, but this must necessarily be considerably modified in details according to the attendant or resulting symptoms.

It is unnecessary in this place once more to review the general management of the patient—this has already been sufficiently indicated in chapter vii. The regulation of light, air, exercise, rest, and sleep is a subject of the greatest importance for the well-being of any one suffering from the secondary effects of a valvular disease, and must be diligently attended to in every case. Dietetic considerations must likewise occupy a first place in the physician's mind, while the excretory channels are carefully watched.

The chief indications for treatment are necessarily based upon the appearances connected with the heart and pulse. The mere existence of a cardiac murmur is of little significance as regards treatment, excepting in this respect, that the conditions are often much more favourable when a loud murmur can be heard than when none is present. Many patients brought under observation in a moribund condition exhibit no cardiac murmur until after recovering to some extent, when the heart, becoming more powerful, is able to generate a murmur. The chief indications to be drawn from the heart are in regard to its size and the energy of its movements and sounds. From the various appearances which are present, it is possible to ascertain whether dilatation or hypertrophy is in the ascendant, and the treatment will thereby be guided.

Of greater importance than the phenomena connected with the heart are the indications yielded by the pulse. When its pressure is low and its rhythm is irregular, it is clear that a remedy which will increase the one and steady the other is in demand.

When the pulse pressure is unduly high, and when this is more particularly associated with arterial sclerosis, the employment of nitro-glycerin, in association with such cardiac remedies as may be necessary, cannot be too highly commended.

Since diminished arterial pressure is the underlying fact in the symptoms of cardiac inadequacy from valvular disease, it necessarily follows that the elevation of blood pressure must be the main object of treatment. This aim is to be achieved by careful attention to all the external surroundings of the

patient, and by the employment of the appropriate cardiac remedies. My own opinions as to the relative value of the great cardiac drugs have been already fully stated, and need not again be referred to, but the difference of opinion as to the utility or otherwise of the digitalis group in aortic disease may be adverted to. It is almost universally allowed that the drugs belonging to this group do not produce such salutary effects in aortic as in mitral disease, and certain writers, as previously mentioned, have practically forbidden the employment of such drugs in aortic incompetence, on the theoretic grounds that by prolonging the period of repose they tend to increase the dilatation of the left ventricle. Such speculative objections have been carried too far, and are merely evidences of a deficiency of philosophic insight. It must indeed be acknowledged by every unbiassed mind that the increased energy of the myocardium far outweighs any fancied effects of the kind mentioned.

The choice of digitalis or strophanthus must be entirely based upon the circumstances of any given case, but, speaking generally, when there is a necessity for rapid action, and if at the same time there be much irritation of the digestive mucous membrane, strophanthus is to be preferred. If, on the other hand, there should be much dropsy and considerable diminution of the renal secretion, digitalis is of greater utility. In the case of digitalis the preparation of most value in most cases is the tincture, but if there be much oedema, the infusion appears to be of greater utility. In certain cases both these fluid preparations are intolerable, when the leaves may be usefully administered in the form of a pill or a powder. The active principles of digitalis are also employed sometimes with beneficial effect. Of these digitalin is preferred for administration by the mouth in the form of Nativelle's granules, while the digitalinum verum of Merck is in many instances of some value hypodermically. Prolonged observations with the former, and considerable experience with the latter, have led me to definite conclusions in regard to both. There can be no doubt of the efficiency of Nativelle's digitalin granules, which produce effects practically identical with the tincture of digitalis. In my own experience the



digitalinum verum is by no means so active as the tincture or infusion, and in spite of the most careful aseptic precautions it has very often set up severe subcutaneous disturbances.

In the case of strophanthus the tincture is the only preparation in common use, but in many instances the employment of strophanthin hypodermically may be resorted to with advantage.

When the cardiac failure of valvular disease is associated with much catarrh of the digestive mucous membranes, the employment of alkalies along with cardiac tonics is advantageous; but in such circumstances the addition of mercury is also of the greatest use. Small doses of blue pill, gray powder, or calomel may be used for this purpose, and it need hardly be said that when such conditions are present without much cedema, the food should largely consist of milk and other fluid substances. In those cases in which there is considerable enlargement of the liver, along with jaundice, more particularly if a tendency to ascites should make itself manifest, the combination of cardiac tonics and mercurial substances is most useful, and the old-fashioned combination of blue pill, digitalis, and squill is admirable.

If the blood is obviously impoverished either in respect of the hæmocytes or hæmoglobin, the addition of iron or of arsenic to the other remedies employed is strongly to be recommended. It is to be remembered, however, that such symptoms are frequently the result of diminished removal of fluid from the system, and that anhydropic methods of treatment may in themselves bring about an approximation to the normal standard.

It is scarcely possible to treat dyspnœa and cyanosis apart from each other. Both may have their origin in simple deficiency of driving power on the part of the heart, from lowered nutrition of the blood, or from some mechanical interference with the aerating surface of the lungs. When these appearances are produced entirely by venous stasis, the usual cardiac remedies will suffice to cause their disappearance, and for both, the use of the special cardiac remedies, along with stimulants both alcoholic and ammoniacal, as well as attention to the general principles of treatment, will obviate



the troubles incidental to them. When alterations in the composition of the blood through deficiency of hæmoglobin produce these symptoms, their effects may be lessened by due attention to nutrition and the use of hæmatinics. In all serious cases marked by those symptoms the hypodermic injection of strychnine and the inhalation of oxygen are of the highest value. When cyanosis is extreme, general bloodletting is certainly necessary, and is often successful in averting disaster. In cases which are desperate the operation of cardiocentesis may even be resorted to. One most interesting and successful case has been narrated by Sloan, who has in connection with it given a very useful summary of the literature of the subject. Through his kindness an opportunity was recently afforded me of examining the patient, who is now in excellent health.

When breathlessness and cyanosis owe their presence to œdematous conditions, the special means for their removal must be such as will be mentioned immediately.

All cases presenting features of œdema show by the presence of this symptom that the hydrostatic conditions are gravely disturbed. For their treatment it is advisable to place the patient in such a posture as to favour the return of blood from the extremities, and to pay due attention to ventilation, while gentle massage is of great utility in assisting the course of the blood. The use of dry diet in such circumstances cannot be overestimated, while the eliminating channels require to be carefully studied. Little doubt is left in my own mind that amongst the cardiac tonics of most use in such conditions the infusion of digitalis is of paramount importance, and it may be that this is due to the fact that it mainly contains digitonin. By combining this preparation with an alkali, more especially with the acetate or citrate of potash, most excellent results may be attained; while other remedies, to be mentioned immediately, which act upon the renal epithelium, may also be employed. At the same time the functions of the skin should be favoured by warmth and such simple diaphoretics as ammonium acetate. The use of saline aperients, more particularly in the concentrated form advocated by Hay, should also find a place in this connection. Of such remedies,

sulphate of soda, or the double tartrate of potash and soda, will be found most serviceable.

When, in spite of remedies acting on the heart, the intestines, the kidneys, and the skin, an œdematous condition persists, or even becomes worse, the transudations, whether subcutaneous or serous, may require removal by mechanical means. For the former Southey's tubes, and for the latter Dieulafoy's aspirator, are most useful.

The renal secretion requires most careful watching, not merely in regard to its quantity, but with reference also to its contents. The quantity is a useful index as to the action of the cardiac tonics. Under the influence of digitalis or strophanthus a diminished secretion usually increases by leaps and bounds, until a certain point has been reached, whence it diminishes again to something like the normal. So long as this is retained the action of such drugs is entirely beneficial; but if the quantity should fall much below this level, it is, as a rule, evidence that the drugs have been employed with sufficient freedom. The amount of urea is helpful as an index of the tissue changes, while the presence of albumin is proof that by stasis the epithelium of the kidney has been damaged.

It is sometimes impossible by means of the cardiac tonics to increase the excretion, and under such circumstances the employment of caffeine may supply the want. My experience has shown that this drug is in every way superior to the combinations of theobromine, commonly sold under attractive names, and there seems little doubt that the inference of Brakenridge as to its direct action upon the renal epithelium is correct.

When sleep is disturbed, as it often is in valvular diseases, it may be possible by simple remedies to secure it. The use of some warm mixture containing alcohol at the hour of repose may sometimes be sufficient, or one of the modern hypnotics, such as paraldehyde, sulphonal, or trional, may bring about the desired result. In many cases, however, no drug save morphine is adequate, and there should be no hesitation about the employment of this remedy, seeing that it has actual stimulant effects upon the circulation.

Such are the general principles which require attention

in the treatment of valvular diseases with failure of compensation. In the most serious cases every means may be necessary. The exhibition of general stimulants, such as alcohol and ammonia, of cardiac tonics, as digitalis or strophanthus, and of nervous excitants, as strychnine, may be imperative; at the same time inhalations of oxygen may be absolutely necessary, massage may be needful in order to effect such results as can only be attained by passive exercise, the excretory channels may require to be acted upon, and hypnotics may be unavoidable. At the same time the diet and the surroundings of the patient must be matters of most careful arrangement.

In cases of less serious import, as well as in those which have to a considerable extent recovered from more grave conditions, the measures applicable to myocardial weakness will be employed. Chief amongst these are careful attention to air, exercise, diet, and surroundings. For such cases the employment of baths and resistance exercises is most useful, and will in most cases lead on to the more active exertion advocated long ago by Stokes, and more recently by Oertel.

## CHAPTER X.

### AFFECTIONS OF THE AORTIC ORIFICE.

IN the overwhelming proportion of cases of aortic disease a double lesion is present, giving rise both to obstruction and incompetence. The reason for this is obvious. Most lesions which produce obstruction of the orifice involve in greater or less degree the cusps, and, by causing contraction or loss of tissue, they interfere with their closure. Aortic obstruction without incompetence is very much more common than incompetence without obstruction, and a moment's consideration is quite sufficient to explain this well-known fact. A certain degree of obstruction may be produced by lesions of the cusps or orifices which do not in any way interfere with the mechanism of the valves; while on the other hand almost every lesion which gives rise to incompetence interferes, more or less, with the exit of blood, and therefore induces obstruction. Incompetence of the valves must be very rarely, if ever, produced by widening of the orifice itself, because from the nature of its construction a stretching of the ring surrounding the origin of the aorta is extremely uncommon.

Statistics bearing upon the relative proportion of cases of pure obstruction and of pure incompetence are highly misleading, inasmuch as in many cases where, as usual, a double lesion has existed, the diagnosis recorded has been that of the affection which appeared to be the more pronounced. It is, nevertheless, of interest to consider the results brought out by Gillespie's analysis of the Royal Infirmary cases during the last five years. The figures are as follows:—Aortic obstruc-



tion, 40 ; incompetence, 141 ; obstruction and incompetence, 230. These results do not accord with my own experience.

### AORTIC OBSTRUCTION.

This disease was observed by Riverius in the year 1646, and was again described by Vieussens shortly afterwards. It was well known to those old pathologists whose observations on chronic valvular disease have been already mentioned. Its recognition during life is of much more recent date. The remarks of Laennec in regard to this affection are by no means definite, and the first clear light thrown upon it is to be found in a very valuable, but unfortunately little known, paper by Hodgkin. Corrigan in a celebrated article, which will be more fully referred to in connection with aortic regurgitation, considers the facts of obstruction. The first edition of Hope's treatise contains rules for its recognition, which are clearly and succinctly laid down ; and from that date the great features of the disease have been well known. Modern additions to our information will be referred to in connection with the description of the special aspect of the disease with which they are associated.

Aortic obstruction may be absolute or relative ; *i.e.*, the orifice may be constricted so as to have less than its normal calibre, or, while the orifice remains of the same diameter as in health, it may, in consequence of the dilatation of the aorta beyond, produce physical signs somewhat similar to those which have their origin in a narrowing of the orifices. This latter condition, however, really belongs to the affections of the aorta itself, and it will be considered in the chapter dealing with its diseases. This section will, therefore, be devoted to obstruction of the aortic orifice from interference with its lumen.

ETIOLOGY.—While aortic obstruction may occur at any age, and in both sexes, it is more frequently found in middle life and advancing years, and it is more common in men than in women.

It is a disease, further, which is often seen amongst those who are engaged in hard physical exertion, and many occupa-

tions, in which long-continued severe muscular efforts are demanded, are especially likely to produce it.

Endocarditis—acute, subacute, and chronic—accounts for



FIG. 137.—Aortic obstruction of vegetative type with incompetence.

a considerable proportion of cases, but it is especially the chronic form of endocarditis which is most prone to lead to aortic obstruction.

Even more important than endocarditis as a cause of aortic obstruction is sclerosis. Nothing need be said here as

to the factors leading to the sclerosis; they have been fully discussed already.

**MORBID ANATOMY.**—The situation of the obstruction may be at the border, or on any part of the arterial aspect of the cusps; at the line of their attachment to the aorta; or at the real origin of the arterial channel, more particularly at that part of the interventricular septum where the aortic cusp of the mitral valve is attached. Vegetations existing at this spot may, as was shown by Chevers, produce the physical



FIG. 138.—Funnel-shaped obstruction of aortic orifice with incompetence.

signs of aortic obstruction, although the cusps themselves are perfectly free from disease.

The nature of the lesion is extremely variable. In these cases which are caused by endocarditis the obstruction may be due to vegetations, as is well shown in Fig. 137. These vegetations differ extremely in size from insignificant granulations to large cauliflower-like masses. They sometimes undergo degenerative changes, or become the seat of calcareous deposition. On the other hand, the valves may be simply thickened along their margins, but this is usually accompanied by a considerable amount of retraction, with degeneration and

calcification. The cusps not infrequently become attached to each other, and this union gives rise to the formation of a greatly diminished aperture occupying, as it were, the summit of a funnel whose apex projects into the aorta. Such a condition is clearly seen in the illustration, Fig. 138, the details of which are given in Case 22, p. 511.

In those cases produced by degeneration, the obstruction is for the most part caused by thickening and sclerosis of the cusps, or of their line of attachment.

Obstruction from injury, whether direct or indirect, is usually produced by one of the cusps floating in the blood current and thus interfering with its outward passage.

The muscular wall of the heart, in cases of pure aortic obstruction, is found to be hypertrophied, without any corresponding degree of dilatation. This condition of matters may exist for a considerable time, but as a rule a certain degree of dilatation ensues. It may in turn lead to incompetence of the mitral cusps, in which case dilatation and hypertrophy of the left auricle ensue, and these in turn may be the starting-point of passive hyperæmia of the lungs and of consecutive changes on the right side of the heart.

The walls of the aorta may be perfectly healthy, but in many cases are the seat of sclerotic changes; when this is the case, there is often dilatation, which may be uniform, but is commonly irregular, giving rise more particularly to bulgings on the concave side of the vessel.

It must not be forgotten, and this will be particularly insisted on in a later portion of the work, that in the sclerotic forms of aortic disease there is apt to be some occlusion of one or both of the coronary arteries, and that when this is the case degenerative changes take place in the myocardium.

**SYMPTOMS.**—In many cases, when there is ample compensation, aortic obstruction gives rise to no subjective symptoms, and the patient has absolutely no feeling of discomfort. Cardiac pain is, however, frequent, and it often presents all the characteristics of angina pectoris, with its different accompaniments. There is sometimes dyspnoea on exertion, along with palpitation.



Aortic obstruction in itself produces but little change in the aspect of the patient, excepting a tendency to pallor, but this, as a rule, is very slight in degree. It must not be forgotten that, as many instances of aortic disease have their origin in, or are connected with, general conditions, the complexion may present features altogether unconnected with the aortic lesion.

The inspection of a patient suffering from aortic obstruction reveals no symptoms connected with the peripheral circulation, unless in those cases in which it is associated with arterial sclerosis. In such instances the more superficial arteries may often be seen to stand out prominently, and to show not only the tortuosity of the vessel, but the characteristic alterations in its axis produced by the pulse wave. Such features are rarely to be observed in the neck.

There is sometimes a perceptible bulging of the præcordia, and distinct pulsations are sometimes to be seen in the third, fourth, and fifth intercostal spaces on the left side. The apex beat is displaced downwards and to the left.

Palpation of the arteries, and particularly of the radial arteries, shows that different conditions of the vessel wall and of the pulse pressure may be present. When there is no considerable sclerosis of the artery, the walls are yielding but elastic; if the lesion is associated with arterial sclerosis the vessel is rigid, and has lost to a greater or lesser degree its elasticity. The blood pressure is also variable, being sometimes above and sometimes below the normal. This fact is easy of explanation. Aortic obstruction in itself produces but little change upon the blood pressure, and this, therefore, depends entirely upon the resistance of the peripheral circulation and the energy of the heart. The pulse is usually infrequent, regular, tardy, and sustained, but it necessarily loses some of these characters in cases manifesting the appearances of cardiac failure. They may also be modified by the super-vention of any intercurrent affection which produces changes in the circulation. Sometimes a thrill may be made out in the larger arteries in aortic obstruction, and this is found to follow the apex beat by an interval longer or shorter according

to the distance of the vessel from the heart. The sphygmographic tracing is often possessed of characters such as are seen in Fig. 139.

When the hand is placed over the præcordia the apex beat is found, as a general rule, to be displaced downwards and outwards so that it may occupy the sixth intercostal space and be situated as far as four or even five inches from mid-sternum. In quality the apex beat is usually strong and sustained, and it is sometimes accompanied by a systolic thrill.

On palpation of the base of the heart, there is sometimes a distinct thrill, absolutely corresponding in time with the apex beat. Sometimes this thrill is conducted, not only over the whole præcordia, but throughout the entire chest; as a

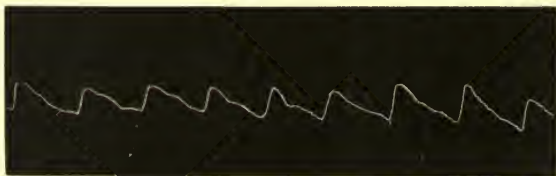


FIG. 139.—Tracing from the radial artery in a case of aortic stenosis; pressure  $2\frac{1}{2}$  oz.

rule, however, it is confined to the manubrium of the sternum and the parts immediately adjacent. It may be present along with such a thrill at the apex as has just been mentioned, the two, however, not being conducted into each other. In this case it seems probable that the thrill at the base is conducted directly from the aorta, while that at the apex is conducted through the left ventricle.

On percussion the area of deep cardiac dulness is almost invariably found to be enlarged, and this enlargement is most distinct towards the left side. The right margin of the heart, in uncomplicated cases, undergoes little alteration in position, but the left margin extends somewhat further outwards, and reaches a point considerably below its usual termination in health. There is, as a rule, no alteration in the extent of the superficial cardiac dulness.

The characteristic feature of aortic obstruction on auscul-

tation is a systolic murmur. In time, the murmur is absolutely synchronous with the apex beat, and although its duration varies considerably, it is usually continued well on towards the second sound. It may, therefore, be represented by the following diagrams (Figs. 140 and 141).

The character of this murmur varies immensely. It may be soft and blowing, or harsh and rasping; while, in some

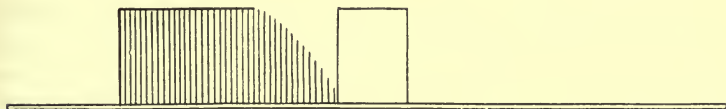


FIG. 140.—Aortic systolic murmur accompanying first sound.

cases, it actually assumes a quality which may fairly be termed musical.

The murmur has its point of maximum intensity above the aortic cartilage, usually about the middle, or even the upper part, of the manubrium sterni. The position of its greatest intensity is, however, extremely variable, although it may be held as a general rule to be above the level of the

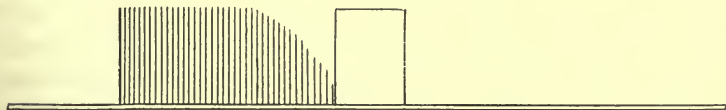


FIG. 141.—Aortic systolic murmur replacing first sound.

conventional aortic area. The murmur may be propagated throughout the whole chest, but, in its most characteristic mode of conduction, it is carried upwards to the summit of the sternum and outwards along the clavicles, as well as downwards by the sternum and ribs to a less extent. It is also, however, conducted along the great arteries. It may be heard up the carotid vessels, and through them may be heard over the skull, outwards by the subclavian arteries, so as to be heard far down the arms, and it may also be conducted by the femoral vessels for a considerable distance down the lower extremities.

It is by no means uncommon for the murmur of aortic obstruction, like the diastolic aortic murmur, to have its greatest intensity to the left of the sternum; and in such cases it is only by the assemblage of the symptoms which accompany it that any certainty can be obtained with regard to the origin of the murmur. This point will be more fully discussed in connection with murmurs generated at the pulmonary orifice.

The aortic second sound presents many different variations, but it is most commonly increased in loudness. It might theoretically be expected to be diminished if the obstruction were excessive; practically, however, this is extremely rare. The first sound in the mitral area is usually of increased intensity and lower tone; it may be roughened in quality, while, when dilatation of the ventricle has occurred, it may be replaced by a murmur. The sounds generated on the right side of the breast are only altered when there are consecutive or attendant changes in the right ventricle.

DIAGNOSIS.—The determination of aortic obstruction does not in most cases present any noteworthy difficulties. The condition of the pulse may vary within such wide limits as to render it of little importance as a guide in the diagnosis of this condition. There may be a total absence of any thrill over the base of the heart, and there may be so little hypertrophy that it hardly reveals itself by any appreciable increase of the cardiac dulness; the sounds, nevertheless, which are generated at the obstructed orifice, are in themselves sufficient in most cases for the establishment of a positive diagnosis. The systolic murmur carried into the vessels of the neck and arms is almost enough to furnish the basis of accurate conclusions.

The only difficulties in regard to the recognition of the lesion arise from the possibility of a confusion between it and a few other affections of the base of the heart or the great blood vessels. The diseases most likely to be mistaken for aortic obstruction, or for which it may be mistaken, are pressure upon the aorta by means of a mediastinal tumour; aneurysmal dilatation of the ascending aorta; obstruction of the pulmonary orifice; and patent ductus arteriosus.



Intrathoracic tumours causing stenosis of the aorta take their origin most commonly from the remains of the thymus gland, or the mediastinal connective tissue. They sometimes, however, arise from some other structures, as, for example, the parietal pericardium or the periosteum of the thoracic walls. Most of these tumours consist in a small cell invasion, and may occur in the course of lymphadenoma or leucocythæmia; they may, however, present the structural features of sarcoma or carcinoma. In other instances the invasion may be by means of tubercular or syphilitic changes. In the case of a mediastinal tumour pressing upon the aorta and causing constriction, there may be, as will be seen later, such definite pulsation from the proximity of the aorta to the anterior chest wall, that the case closely resembles an aneurysm. But in other instances, and these are precisely the cases which require such reference in this place, there may be no apparent pulsation, and yet on auscultation the systolic murmur is heard over the manubrium of the sternum, and is often carried along the great arteries. In such a case there is an area of dulness on percussion corresponding to the invasion, which in itself is sufficient to differentiate the condition from simple obstruction of the aortic orifice. There is, in addition, no necessary alteration in the structure of the arteries throughout the body, and there is almost invariably no alteration in the character of the second sound in the aortic area.

Aneurysmal dilatation of the ascending aorta will be more fully considered in a later portion of this work, but a passing reference to it is necessary at this point, in order to show how it may be differentiated from obstruction at the aortic orifice. The only form of dilatation at all likely to be mistaken for aortic obstruction is the fusiform type, inasmuch as the sacculated variety is most unlikely to cause any difficulty on account of the well-marked physical signs to which it gives rise. Simple fusiform dilatation of the aorta reveals itself by three physical signs, one of which, however, is frequently wanting. On mapping out the cardiac dulness in this condition, it is not infrequently found to extend further outwards to the right than usual at the level of the second and third

costal cartilages. This is the physical sign which is most likely to be absent, and when this is the case there remains only the evidence furnished by the ear. On auscultation, there are two important physical signs in simple dilatation of the aorta. There is, in the first place, the soft blowing systolic murmur produced by eddies which take their origin within the dilated aorta; for it must be remembered that a dilatation of that vessel gives rise to practically the same physical conditions as are present when there is an obstruction of the aortic orifice. There is, in both instances, a wider channel beyond a narrower orifice, through which the blood is driven. In this way, dilatation of the ascending aorta comes to present what may be termed relative obstruction of the aortic orifice. The murmur so produced is carried along the vessels of the neck and of the arms precisely as is the case in aortic obstruction, and in itself, therefore, the murmur furnishes no criterion as to whether there is absolute, or only relative, obstruction. The other physical sign furnished by auscultation is accentuation of the aortic second sound, caused by the greater column of blood falling backwards upon the aortic cusps. This furnishes, on the whole, the most important evidence of dilatation of the ascending aorta. There is, nevertheless, a combination of circumstances in which accentuation of the aortic second sound may be present without any dilatation of the aorta. In arterial sclerosis, more particularly when accompanied, as is often the case, with chronic cirrhotic changes in the kidneys, the aortic second sound may be very greatly accentuated. It must be remembered, moreover, that in such a combination of circumstances there is very frequently some endocardial mischief about the aortic cusps, and that here, therefore, there may be a systolic aortic murmur along with accentuation of the second sound. In such a condition, and it is one far from uncommon, the second sound has not the drum-like character, and there are also, as a rule, other appearances to guide the observer to correct conclusions. The presence of albuminuria, of diminished urea secretion, and of albuminuric retinitis, as well as the peculiar characters of the pulse, may render valuable service in this respect.

Pulmonary obstruction is occasionally simulated by the disease now under discussion. The characteristic systolic murmur in aortic disease may be heard, as already mentioned, with its greatest intensity to the left of the sternum, and when this is the case, it may readily be mistaken for a pulmonary systolic murmur. The pulmonary systolic murmur can never, by any possibility, be propagated into the great arteries at the root of the neck, and this alone is sufficient to prevent any confusion between the two affections.

Patent ductus arteriosus, as has been fully shown when the congenital affections of the heart were under consideration, is characterised by a loud systolic murmur, heard with its greatest intensity in the second left intercostal space about one inch and a half to the left of mid-sternum. This murmur may be propagated, not only throughout the whole upper part of the chest, but, indeed, throughout the whole body, as is also the case with many murmurs produced by aortic obstruction. The great points of difference between the auscultatory phenomena in these two conditions are that the murmur has its maximum intensity in the position just indicated, and that it is late systolic in rhythm, while it frequently, if not usually, continues beyond the second sound. It is, however, never carried into the vessels of the neck or of the arms. It may seem unlikely that this condition could ever be mistaken for aortic obstruction, but one of the cases described has been more than once diagnosed as an instance of aortic obstruction.

PROGNOSIS.—Aortic obstruction is, in itself, less serious than any other organic valvular disease. Free from many of the disturbing clinical features common to other valvular lesions, it has, moreover, not the tendency to sudden death manifested by aortic regurgitation, and its duration is more prolonged than is the case in the other organic valvular affections. These considerations, however, must be qualified by the remembrance that the lesions of aortic obstruction are apt, in common with all pathological alterations of the orifice and its valves, to interfere with the coronary arteries. When this is the case there may be distressing symptoms, and even sudden death. This fact of sudden death in cases of uncomplicated

aortic obstruction has recently been the subject of an interesting communication by Kelynack.

**TREATMENT.**—There is, as a rule, in this disease no need for special treatment, and attention to general principles is all that is necessary.

In the group of cases taking their origin in chronic sclerotic processes, it is probable that the course of the lesions may be, to some extent, diminished by the careful regulation of habits as to food and drink, exercise and rest. The use of food which is not too highly nitrogenous, and the employment of ample diluents, must certainly be recommended. All physical and mental stress must be, as far as possible, eliminated, although sufficient exercise must be enjoined in order to assist the metabolic processes of the body.

It is probable that the use of iodide of potassium retards the sclerotic process, and its long-continued use in moderate doses should certainly be tried. When cardiac failure ensues, as it is at last apt to do, the treatment must be conducted upon the general principles which have already been laid down.

**CASE 17.**—J. O'B., aged 64, widower, itinerant photographer, was under my care in the Royal Infirmary suffering from breathlessness. The patient's father died at an advanced age of apoplexy. His mother lived to a considerably greater age, and died, according to the patient's account, from decay. A brother and two sisters always enjoyed good health; one brother was killed by an accident, and one sister died from a cause unknown. The patient's social conditions had been satisfactory, but his mode of life had not been conducive to health.

He had suffered during the previous ten or eleven years from bronchitis during each succeeding winter, but had not had any other ailment, with the exception of the usual children's diseases in youth.

The present illness began in the year preceding admission, with breathlessness. There was never any swelling of the ankles.

There were no symptoms connected with the digestive organs, but the tongue was coated and fissured. The glandular system presented no evidence of disorder. The superficial arteries were rigid and tortuous. The radial pulse was full and of high pressure. The pulsation was 61 per minute and perfectly regular. Each individual pulsation was tardy and sustained. There were no abnormal appearances connected with the arteries or veins of the neck. Some epigastric pulsation was present. The apex beat was distinctly to be seen in the fifth intercostal space in the mammillary line. On palpation, the apex beat was felt to be diffused. The area of cardiac dulness extended two and four inches respectively to



the right and left of the mid-sternum. On auscultation, a loud and somewhat harsh systolic murmur was heard over the whole præcordia. Its maximum intensity was over the upper part of the manubrium sterni, and it was propagated over the whole of the præcordia, into the left axilla, and as far as the left scapular angle behind. It was also conducted up the carotids, and could be heard on auscultation up to the vertex of the skull as well as along the subclavian arteries, so that it could be heard as far as the wrist, and down the femorals as far as the knees. The second sound, although loud, was not tympanic. A few sonorous rhonchi could be heard over the chest.

The urinary secretion was considerably increased, the average amount being from seventy to eighty ounces per day.

Under treatment with iodide of potash and rest he greatly improved.

This case is an excellent example of uncomplicated aortic obstruction obviously due to degenerative changes, and accompanied by renal cirrhosis.

CASE 18. *Aortic Obstruction*.—K. C., aged 21, unmarried, housemaid, came under observation in the Royal Infirmary, 29th June 1893, complaining of a choking sensation, and general shaking over the whole body. Her father had died at the age of 45 of some liver affection; her mother at the same age of consumption. All her brothers and sisters, three and two in number respectively, had enjoyed excellent health. The only affection from which the patient had suffered in the past was a severe attack of measles, which was followed by abscess of the jaw. The attack for which she sought advice began quite insidiously. She had undergone somewhat arduous labour for a considerable period and found herself becoming gradually weaker.

The patient's general aspect was quite healthy. Her lips were ruddy, and her cheeks bright. Her teeth were much decayed; the tongue was furred, and there were complaints of weak digestion. The radial walls were perfectly healthy, and the vessels full with rather more than average pressure. The pulse varied slightly from day to day, but was usually a little below or above 80. It was somewhat tardy and sustained, but absolutely regular and equal. On inspection of the neck and chest, no abnormal appearances could be detected. There was no visible pulsation of the vessels of the neck, and no abnormal movements over the præcordia. Palpation revealed that the apex beat was in the fifth intercostal space two and three-quarter inches from mid-sternum. No thrill could be felt in any part of the chest. By percussion, the right and left borders at the level of the fourth costal cartilages were found two and three inches from mid-sternum. On auscultation, a distinct systolic murmur was heard over the base of the heart. Somewhat harsh in character, its maximum intensity was at the attachment of the first right rib to the sternum, from which point it was conducted to some extent in every direction, but more particularly along the right clavicle, and the parts immediately below it, as well as up both carotid arteries. The second sound in the aortic area was accentuated.

ated, being much louder than the second sound in the pulmonary area. It was also frequently doubled, a fact which could also be ascertained in the pulmonary region.

The first sound in the mitral and tricuspid regions was clear and distinct. It could, in fact, be heard with perfect distinctness over the whole præcordia, being obviously only accompanied at the base by the murmur which more or less obscured it. There were no symptoms of disturbance connected with the respiratory or any other system.

It was clear that in this case there was simple aortic obstruction, without any regurgitation. The only point of difficulty was the determination of the nature of the obstruction, whether it was to be regarded as absolute, from some change in the orifice or its cusps, or as a relative obstruction, produced by dilatation of the aorta beyond the orifice. The tardy pulse was strong evidence in favour of the former view, while the great accentuation of the aortic second sound was a powerful argument in favour of the latter. Taking all the facts into consideration, it seemed probable that there was a combination of organic change in the orifice along with some dilatation of the aorta. The origin of such lesions was necessarily a matter of pure speculation, but it seemed probable that endocarditis was the potent factor, associated in all probability with severe physical stress.

By means of absolute rest and careful diet, along with the use of strophanthus and nux vomica, the patient recovered from the symptoms for which she came under treatment, and although she necessarily left the Infirmary with the physical signs unaltered, her general condition had greatly improved.

### AORTIC INCOMPETENCE.

In consequence of his views on the second sound, it was of necessity an impossibility for Laennec to arrive at any diagnosis of this disease, and its discovery was reserved for some of his immediate successors. Hodgkin, in the paper which has already been referred to, published, in 1829, the first clinical description of the disease. It is somewhat singular that for many years his observations escaped general notice; in fact, until the publication of a note

on the history of valvular diseases by Wilks, they appear to have attracted no attention. In his paper Hodgkin describes, amongst others, the case of a medical friend who suffered from very violent, although perfectly regular, arterial action, with a thrill in the arteries, and great throbbing of the carotids. But he goes further than this, because he describes a murmur which, he says, "presented this peculiarity, that it was double, attending the systole as well as the diastole." These facts show that Hodgkin had fully grasped the most important clinical features of aortic incompetence, and from the pathological point of view, as has been mentioned previously, as well as from the standpoint of etiology, his important paper is well worthy of perusal. It is of great interest to observe that he attributes the aortic lesions in some cases to excessive muscular effort.

Corrigan has almost universally had the credit of being the first to describe the symptoms and to connect them with the lesions of aortic obstruction, but his paper was published three years later than that of Hodgkin. It must be noticed also that while his descriptions are in most points lucid and complete, there is nevertheless some little ambiguity as regards the murmurs produced by aortic incompetence. In the year which witnessed the publication of Corrigan's celebrated paper, appeared the classical work of Hope, in which the diastolic murmur of aortic incompetence was, for the first time, incorporated in a systematic treatise. The further additions to our knowledge of aortic obstruction will be described as they emerge in the sequel.

Aortic incompetence is extremely rare as an isolated lesion, but from the statistics which have been already referred to it is in association with obstruction of the orifice one of the most frequently observed cardiac diseases. The reason for this is not difficult to find. The aortic orifice is supported by an extremely resistant fibrous ring which seems almost incapable of dilatation under ordinary circumstances, so that regurgitation from the aorta into the left ventricle can only take place by means of absolute incompetence of the cusps, not through any increase in the size of the orifice.

ETIOLOGY.—The causes which produce incompetence of the

aortic cusps are practically those which have been already mentioned as leading to obstruction of the orifice.

Endocarditis arising in consequence of any of the factors which have been previously discussed may give rise to lesions of the cusps resulting in their inadequacy. There can be no doubt, as is indeed generally allowed, that endocarditis does not play such an important part in the evolution of aortic as of mitral diseases, and when this form of valvular lesion takes place it is found most commonly in association with similar changes in the mitral valve.

The incompetence may have its origin, as is certainly more commonly the case, in sclerotic changes, produced, as has been mentioned before, by long-continued stress, although there cannot be a doubt that certain toxic agents, of which alcohol may be taken as a type, play an important part also in the evolution of this more chronic form.

The association of this cardiac lesion with locomotor ataxia, first noted by Vulpian, and Berger, and Rosenbach, has led some writers, and more particularly certain members of the modern French school, to theorise upon the possible mode in which they are connected. Grasset, for instance, is of opinion that a reflex dilatation of the heart produced by the influence of the pain may lead to the aortic lesion, a view which must to an ordinary intelligence appear somewhat fanciful; Wood is inclined to regard the aortic lesions as resulting from trophic lesions produced by the affection of the posterior nerve roots; Letulle and Martin believes that the spinal lesions and the aortic changes are due to the same cause, endarteritis obliterans, and this view is not without weighty arguments in its favour; Raymond considers that the association is purely accidental and that the two lesions are not directly connected with each other.

Aortic incompetence is the most frequent cardiac lesion produced by injury. This has been particularly referred to in connection with the etiology of valvular affections in general, and the only point requiring further reference in this place is that the damage, as was first pointed by Foster, is usually confined to the left posterior cusp.

**MORBID ANATOMY.**—Incompetence of the aortic valve, as tested by its capability of retaining fluid, is for practical



purposes to be regarded as always brought about by an affection of the cusps themselves. The possibility, however, of regurgitation by means of a dilatation of the orifice rendering it too wide to be closed by the cusps must theoretically be granted. It was admitted by Corrigan in his original paper and has been a subject of discussion ever since, more particularly in France. Suffice it to say that this conjecture has never been proved, and that it must for the present remain merely a speculative possibility. The most frequently observed lesions permitting of regurgitation are due to sclerotic processes, in consequence of which there is shrinkage and deformation of the cusps, along with thickening. These are frequently associated with changes in the walls of the aorta, such as patches of sclerosis, atheroma, and calcification, often along with dilatation of the aorta beyond the orifice, which may be either general or partial. Frequently associated with such changes are alterations at the openings of the coronary arteries, which are very apt to be obstructed in consequence of the lesions of the aortic walls.

Next in point of frequency are the alterations of the cusps which take their origin in some form of endocarditis. The lesions which are produced in this way may be vegetative, in which case a number of vegetations of extremely variable size and consistence are found upon that side of the cusps opposed to the blood current. These are illustrated by Fig. 137 on page 474. Very frequently in this case there is some loss of substance in consequence of ulcerative change, and perhaps still more commonly there are deformities produced by retraction of the cusps. It need hardly be added that in old-standing instances of disease there are often calcareous deposits producing a very considerable amount of rigidity.

Instead of vegetations the endocarditic process appears in some instances to have a tendency rather to produce a fusion of the cusps at their free edges, so as to bring about the production of a small aperture often of a curious form. In this case also the growth of fibrous tissue and the deposit of inorganic salts may lead to excessive hardness and rigidity. Fig. 138, p. 475, furnishes a capital example of this.

It must be obvious that in all these conditions the result

upon the circulation must be to produce at once obstruction of the orifice and incompetence of the cusps, and it therefore follows, as was mentioned above, that aortic incompetence is extremely rare except in combination with obstruction.

The effects of aortic incompetence are felt in the first place by the left ventricle. As a result of the escape of blood with each diastole, there is an increased amount of blood in the ventricle during that phase, the consequence of which is that the ventricle becomes surcharged. It has accordingly an increased amount of work to do, as well as a larger amount of blood to accommodate. The wall becomes hypertrophied in order to meet the one requirement; the cavity becomes dilated for the purpose of adapting itself to the other. So long as the nutritive possibilities of the coronary arteries are not impaired, and the character of the blood is such as to afford an adequate supply of material to meet the metabolic requirements of the myocardium, the process of hypertrophy provides for every contingency, and the process of compensation leads to perfect equilibrium. In one or two ways, however, the balance may be upset. The process of hypertrophy may in itself defeat the ends which it is obviously intended to subserve, because the heart may outgrow the possibilities afforded by the coronary arteries to maintain its nutrition. It will be more fully mentioned in the sequel that hypertrophy is very commonly associated with degenerative changes apparently even from the first, and when the coronary arteries are not competent to support the muscular wall, some form of degeneration is inevitable. From that moment dilatation is in excess of hypertrophy, and cardiac failure is the result. On the other hand, in consequence of the severe strain to which it is subjected, the mitral orifice may become dilated, or its cusps may be damaged. So long as there is integrity of these cusps there is no implication of the circulation in the lungs, and the right side of the heart is not interfered with. It is far otherwise when the mitral cusps fail to close the left auriculo-ventricular orifice. When this happens, there is backward pressure upon the left auricle, the pulmonary veins, and the lung tissue, as a result of which passive venous hyperæmia takes place, entailing all its consequences upon

the right side of the heart, and, through it, upon the general venous circulation.

In aortic incompetence, moreover, it must not be forgotten that the regurgitation of blood leads to troubles connected with the periphery of the arterial circulation. The interference with the integrity of the cusps leads to an excessive difference in arterial pressure during cardiac systole and diastole, and its swift fall at the commencement of the diastole causes the flow in the arterial system to become less continuous. It therefore results that the larger vessels near the heart are subject to enormous variations in blood pressure, while those which are far away are not so thoroughly supplied with blood as in health. It may further be remarked in this place that these alterations, affecting as they do the coronary arteries, tend to lessen their nutritive possibilities, and if these coronary vessels should be affected by arterio-sclerosis, the interference with the nutrition of the heart must be greatly increased.

SYMPTOMS.—Aortic regurgitation gives rise, as a general rule, to very few symptoms so long as compensation preserves the equilibrium of the circulation. The only exceptions to this statement are furnished by those cases in which one or more of the cusps have been ruptured by traumatism, or have undergone very rapid destructive changes in consequence of grave endocarditis. In such cases as these, the onset of general symptoms may be almost dramatic. The rupture of a valve in consequence of injury or strain may at once lead to severe pain and dyspnoea, along with disturbances to the circulation.

Such instances as these are of rare occurrence. An aortic incompetence remains, in the overwhelming proportion of cases, absolutely latent for a considerable period. Examples proving the correctness of this statement frequently present themselves before every physician. One of the most striking which has come under my own notice was the case of a friend between forty and fifty years of age, with whom it was my custom to spend some time every year. On one occasion, after a forty-mile walk, embracing the ascent of Cader Idris, he informed me that he felt a little uneasiness and palpitation,

and would like me to auscultate his chest. On doing so there were, to my consternation, all the evidences of serious aortic incompetence. With the avoidance of all severe physical exertion for the future, my friend lived with every appearance of excellent health for some years, but, having one day somewhat imprudently hurried to catch a train, he fell back dead a few minutes after being seated in it. In this instance, as in so many others, the heart was quite able by means of perfect compensation to overtake all ordinary work, but our forty-mile walk had proved, unfortunately, too much for it.

Even where compensation appears to be adequate, there are sometimes symptoms of disturbance. During repose there may be nothing to direct attention to the circulation, while on any exertion there is palpitation or fluttering of the heart. Breathlessness on exertion may also make its appearance, and sometimes attacks of this kind may really deserve the term of cardiac asthma. Certain of the instances in which such an occurrence takes place are apparently due simply to the mal-aeration of the blood through interference with its return. In other cases, however, they are associated with changes in the kidney which have taken place along with arterio-sclerosis. The fact, further, must not be overlooked that some patients presenting this prominent symptom have morbid changes in the lungs, interfering with the area of aerating surface. Petit lays considerable stress upon symptoms of painful dyspepsia, and such accompaniments must be admitted as not altogether uncommon. They make their appearance, as a rule, after excessive exertion.

Cardiac pain is one of the commonest symptoms, and may present all degrees of intensity, from very slight uneasiness to the most profound agony. Such painful sensations are fully dealt with elsewhere, and the subject requires no further comment in this place. Patients who suffer from aortic incompetence are not infrequently liable to various symptoms connected with the cerebral functions. Besides the sensation of throbbing, which is often present, there may be headache and giddiness, both of which are more conspicuously present on exertion, and are frequently attended also by subjective



sensations connected with the ears, and less commonly also with the eyes. There may be sleeplessness and disturbance of the higher faculties. One and all of these are due to the disturbance of the peripheral circulation within the cranium.

There is, as a rule, pallor of the surface, produced by the disturbance of the peripheral circulation; this arises sometimes entirely in consequence of the valvular defect, but sometimes is due also in part to sclerosis of the arteries.

On examining the behaviour of the peripheral circulation there are certain important appearances of common occurrence. On any part of the skin normally pale, which has been rendered red by pressure or by friction, there may be seen what is known as the capillary pulse. This is an increase of the redness following each systole of the heart, and a paleness with every diastole. The causation of this is sufficiently obvious. It is due to the relatively unfilled condition of the peripheral vessels allowing of great oscillations of the blood pressure. This symptom appears to have been first described by Quinke, and it has been the subject of exhaustive observations in later times. The same alternating flushing and paling of the surface is often present, as was pointed out by Hirtz, in the halo surrounding the patch of urticaria when this occurs in a patient suffering from aortic incompetence. A similar appearance may be detected close to the lunules at the base of the nails. It is best exhibited when the free border of the nail is slightly pressed downwards. On examining the fundus of the eye with the ophthalmoscope, increased pulsation of the arteries may be observed. Müller called attention to pulsatory movements and swelling of the palate, uvula, and tonsils. Balfour does not admit that these appearances are of any value in diagnosis, and it must be allowed that they may occur in conditions permitting a free passage of blood through the arterioles and capillaries into the veins. In point of fact the capillary pulse is, as Whittaker remarks, an indication of the force of the left ventricle.

The most characteristic appearance presented by any patient suffering from aortic incompetence is the excessive pulsation of all the arteries. This is without doubt best

exhibited by the carotid arteries in the neck, which are seen by their violent throbbing to produce extensive movements of the neck; but branches of the facial and temporal arteries may be seen to throb violently, while the subclavian and brachial arteries also show excessive movements. In the case of male patients with beards or whiskers, these appendages are seen to execute lively movements, and the pulsation is in some instances so violent as to give rise to distinct movements of the head. It need hardly be added that the superficial arteries of the lower limbs also show the same exaggerated pulsation. The radial pulse on palpation shows those striking characters summed up under the term Corrigan's pulse. Corrigan did indeed give an excellent description of these features, but, as has been mentioned above, they were studied previously by Hodgkin in full knowledge of their connection with this disease. The essential points, indeed, were noticed long ago by Vieussens. The principal features of this pulse are that the vessel is far from full in general, yet the pressure is often above the normal. The pulse may be frequent or rare according to circumstances, but the rhythm is, in the great proportion of cases, perfectly regular, and the pulsations are equal or nearly so. These characteristics undergo modification when cardiac failure comes on. Each individual pulsation is abrupt and large, but it is of short duration, falling away from the fingers in the instant when it reaches them. It gives the feeling to the fingers laid lightly upon the artery as if a small shot had touched them and had instantly recoiled. From its giving the fingers this sensation, it has been well termed the water-hammer pulse, since the sensation which it produces is remarkably like that of the throbbing caused by the vibration of that toy.

A sphygmographic tracing of the pulse and the radial artery brings out some of these points very distinctly. The ascent of the curve is abrupt and steep, rising to a higher level than in almost any other condition. The descent is almost as swift, and there may be a considerable tidal wave, but the most remarkable feature is that the dicrotic notch and wave are very slightly marked. These characters are shown in Fig. 142.

There is one appearance sometimes, but not very commonly, seen in cases of aortic incompetence—the development of a direct venous pulse in the peripheral veins. This is a wave passing from the periphery towards the centre, and distinctly following the systole of the ventricle. It is best seen in the veins upon the back of the hand, but it may be seen in other superficial veins, and it is a gentle undulation. Sometimes there is sufficient movement to allow of a tracing, as will be seen in Case 20, narrated below (p. 506).

The examination of the front of the chest shows in general no alteration in form, except where a small chest is associated with considerable enlargement of the heart. It

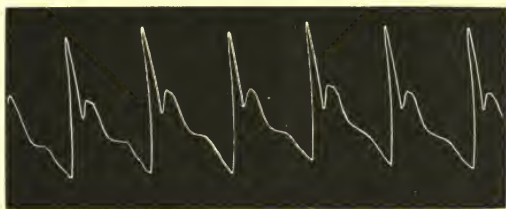


FIG. 142.—Tracing from the radial artery in a case of aortic incompetence ; pressure 3 oz.

may happen in such a combination of circumstances that there is a considerable amount of bulging forward, but apart from this there is no abnormal appearance. In most instances the apex beat occupies a position farther down and to the left than in health. In point of fact, it is extremely common to see it in the sixth left intercostal space quite outside the mammillary line. Its position cannot always be determined by simple inspection, as it may be diffuse, as is more particularly the case when it impinges upon the sixth rib. That the cardiac systole is considerably augmented in force can usually be seen from the distinct movements of the præcordia—movements which at once occupy a wider area, and are characterised by greater amplitude.

The application of the hand at once reveals the fact of increased force of pulsation. It shows, moreover, that not merely is the cardiac systole more powerful, but that the diastolic recoil is increased. Palpation further brings out

the exact position of the apex beat, and proves that its force is increased. But there is another sensation communicated to the hand at the apex, a sensation as if the apex beat were accompanied by something like a thrill. This is the result of the greatly increased energy of the heart, resulting from its hypertrophy. There is occasionally a distinct thrill felt, more especially in the superior sternal region, which accompanies the diastole. It may be felt over the whole præcordia, and very distinctly even at the apex of the heart. This thrill when present is due to vibrations produced by the escape of the blood backwards from the aorta.

The area of cardiac dulness, as ascertained by percussion, is always increased, and the form which it assumes is lengthened in the vertical direction as well as widened horizontally. The right border of the heart is not so much displaced outwards to the right as is the left in the opposite direction, and the most characteristic change is that the cardiac dulness is found beyond the fifth intercostal space as far as the sixth; sometimes indeed it reaches the seventh rib.

Auscultation reveals the characteristic diastolic murmur. The character of this murmur has wide limits of variability. It often has a perfectly soft blowing sound, but it may be harsh and rasping in quality, and it is above all others that which most frequently assumes the character of a musical sound. These differences must of necessity depend upon the size of the orifice through which escape is allowed, the nature of its borders, and the arterial pressure. The harshest murmurs are those which have their origin in those cases where a cusp has been ruptured, or in which there has been some loss of texture.

The position of maximum intensity of this aortic murmur is also subject to considerable variation, but it may be laid down as a general principle that its maximum intensity is very rarely in the conventional aortic area. In the majority of cases it is heard most distinctly over the right half of the sternum, about the level of the fourth costal cartilage; it is not an uncommon experience to find that it is most distinctly heard to the left of the sternum altogether, about the third costal cartilage or third intercostal space; it may sometimes



be heard only at the apex of the heart and nowhere else. The explanation of these divergent results is at present impossible. They can only be attributed to individual peculiarities lying beyond the reach of observation.

The diastolic aortic murmur is for the most part conducted downwards in the direction of the xiphisternum and of the apex of the heart. The extent of its propagation has some relation to the harshness of the murmur—a rule which is not, however, without some exceptions. The soft blowing type of murmur is as a rule not heard farther than the junction of the body of the sternum with the xiphoid cartilage, and it very often fails to reach the apex of the heart. A harsh or musical murmur, on the other hand, may not only be heard throughout the whole chest, but it may even be heard by a bystander without the necessity of applying his ear to the chest at all. In those cases where the diastolic murmur is distinctly heard at the apex, there is, according to Foster, a lesion of the left posterior aortic cusp.

About thirty-five years ago Flint called attention to a discrepancy between the results of clinical observation and pathological investigation as exemplified in two cases under his own care. In each of the two patients there was a presystolic murmur, which in both possessed a blubbery character. After death, in the first case, it was found that “the aorta was atheromatous and dilated so as to render the valvular segments evidently insufficient. The mitral valve presented nothing abnormal, save a few small vegetations at the base of the curtains as seen from the auricular aspect of the orifice.” After death, the post-mortem examination in the second case showed that “the aortic segments were contracted, and evidently insufficient. The mitral curtains presented no lesions; the mitral orifice was neither contracted nor dilated, and the valve was evidently sufficient.”

Flint attempted to account for the discrepancy in the following way:—The explanation involves a point connected with the physiological action of the auricular valves. Experiments show that when the ventricles are filled with a liquid, the valvular curtains are floated away from the ventricular sides, approximating to each other and tending to closure of the

auricular orifice. In fact, as first shown by Baumgarten and Hamernjk, of Germany, a forcible injection of liquid into the left ventricle through the auricular opening will cause a complete closure of this opening by the coaptation of the mitral curtains, so that these authors contend that the natural closure of the auricular orifices is effected, not by the contraction of the ventricles, but by the forcible current of blood propelled into the ventricles by the auricles. However this may be, that the mitral curtains are floated out and brought into apposition to each other by simply distending the ventricular cavity with liquid, is a fact sufficiently established and easily verified. Now in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta as well as from the auricle, before the auricular contraction takes place. The distension of the ventricle is such that the mitral curtains are brought into coaptation, and when the auricular contraction takes place the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbering murmur. The physical condition is in effect analogous to contraction of the mitral orifice from an adhesion of the curtains at their sides, the latter condition, as clinical observation abundantly proves, giving rise to a mitral direct murmur of a similar character."

Charlewood Turner narrated two cases in which a pre-systolic murmur was associated with aortic incompetence, and in which no mitral lesion was found on post-mortem examination. In the same communication he placed on record another case in which a presystolic murmur attended by a thrill was heard distinctly during the life of the patient, and in which on post-mortem examination the condition of the orifices and valves was perfectly healthy, with the exception of a small patch of atheroma on the anterior mitral cusp adjacent to the aortic valve. The aortic cusps were perfectly normal. Turner maintained the presystolic murmur to be the result of regurgitation, he himself being a stout champion of Barclay's views alluded to in another chapter.

Guitéras narrated an instance of aortic disease in which, with the diastolic murmur of aortic regurgitation, there was a

presystolic murmur at the apex of the heart running up to a weak first sound, and in which on post-mortem examination there was besides the aortic lesion only a little thickening of the base of the mitral cusps without any change in their edges. The same writer in a subsequent article described another case in which with similar appearances there was a total absence of any mitral lesion. He expresses the opinion that obstructive functional mitral murmurs frequently occur in aortic regurgitation, more especially when the posterior aortic segment is affected, and explains them by the direction of the regurgitant stream which is brought to bear directly against the anterior cusp of the mitral valve.

Osler placed two cases on record in which, along with aortic disease, there was a presystolic mitral murmur with integrity of the mitral cusps on post-mortem examination.

Steell on one occasion found a healthy mitral valve in a case of aortic disease presenting symptoms of mitral obstruction.

Bramwell published an instance in which, with all the evidences of aortic incompetence, there was a presystolic murmur at the apex along with, however, a systolic murmur conducted to the scapula, and in which from cardiographic tracings he concluded that probably there was no mitral obstruction. As no post-mortem examination was obtained, this case is bereft of the last link in the evidence required. He is of opinion that the occurrence of the presystolic murmur "may perhaps be explained by supposing that in those rare cases in which it is met with, the lesion chiefly affects the posterior coronary segment of the aortic valve, with the result that the full force of the regurgitant current falls, as it were, upon the great anterior segment of the mitral valve, forcing it into the position which Flint has described as essential for the production of the murmur." This is identically the same explanation as that advanced by Guitéras.

Gairdner recorded a very interesting case in which an auriculo-systolic murmur was heard, and in which there was an aperture in the right curtain of the aortic valve, about half an inch in diameter and with the upper part about one-eighth of an inch from the edge of the curtain. The aperture was surrounded by lobulated projections of a white colour. In

this instance by inference the mitral cusps were healthy, although no express statement is given in regard to this point.

Maguire has very carefully recorded two cases quite analogous to those, in one of which there was only a little thickening of the mitral cusps, and in the other only a few scattered patches of atheroma on the anterior mitral cusp. In a careful review of the opinions of previous writers, and of the eleven cases placed on record up to the appearance of his paper, he expresses the view that in aortic regurgitation the anterior mitral cusp during diastole lies between two blood streams, irregularities in which would readily produce vibrations in the cusp. When the aortic regurgitant stream is very copious a diastolic murmur may be heard, as in mitral obstruction, near the apex of the heart, and actually produced by the mitral curtain, not merely conducted by it from the aortic orifice; further, that, when the contraction of the auricle increases the rate of the stream coming through the auriculo-ventricular opening, a presystolic murmur might be produced. Maguire very justly criticises the theory of Flint on account of his misapprehension of the mechanism of the cusps, for as has been already fully mentioned in the anatomical introduction, the mitral cusps hang vertically from the mitral orifice during diastole, and there is no such phenomenon as the floating-up which he described.

Lees described four cases of a similar character to those above referred to, and sees no difficulty in accepting the view that the mitral cusp is thrown into vibrations by two independent blood currents impinging on its opposite sides.

Lespérance fully entered into the question, and concluded that the murmur is produced by the presystolic shock of the heart upon the layer of lung in front of the heart.

Sansom is of opinion that, if the mitral cusps are brought nearly together without completely closing the orifice, there is a possibility of the presystolic murmur, and allows that there may be two explanations:—The force of the current impinging upon the lower surface of the anterior mitral cusp might, by obstructing the stream from the auricle, produce a *de facto* impediment at the end of each diastole, or vibrations might



be directly communicated by the aortic regurgitant stream to the mitral cusp.

In the contribution of Potain he accepts the explanation that the presystolic murmur has its origin in vibrations of the great cusp of the mitral valve, produced by the two currents which impinge upon its surfaces.

Being thoroughly conversant with Flint's views since the appearance of his second paper, the points under discussion have been diligently sought for by me; so far, however, without result. Cases have frequently presented themselves no doubt in which, with absolute evidence of aortic disease, there has been a presystolic murmur, but in every one of these, without exception, post-mortem examination has revealed mitral obstruction as well as aortic lesions.

The observations of so many able physicians have raised this subject beyond all possibility of doubt, and the only element of uncertainty surrounding it is as regards its causation. The opinion expressed by Flint is of course absolutely untenable. No one with any knowledge of the physiology of the intracardiac movements could for one moment uphold it. The other explanation, which has been advanced by Guit  ras and accepted by subsequent writers, may very well be accepted as a valid explanation of the facts unless an even simpler supposition might serve the purpose,—that of the mingling of the two currents in the neighbourhood of the apex of the heart.

Duroziez directed attention to a phenomenon ascertained on auscultation of the arteries in aortic incompetence. On auscultation of an artery with the circulation in a condition of integrity, one sound is usually heard; occasionally there may be two. If the stethoscope is firmly pressed upon an artery in this condition a distinct murmur will be heard immediately following the ventricular systole, and if there have previously been two arterial sounds, this murmur will be followed by the second of these. In cases of free aortic regurgitation two murmurs are heard, one immediately succeeding the systole of the ventricles, the other immediately following their diastole. These murmurs are of local origin, and are produced by the narrowing of the lumen

from pressure of the stethoscope. The first of these two murmurs accompanies the wave of increased pressure during the ventricular systole, and may be produced in all conditions. The second can only be heard when there is a backward current towards the heart in free aortic regurgitation. Duroziez allowed that the double murmur might be heard in conditions other than those of aortic regurgitation, but Balfour holds that "a true ventricular-diastolic murmur audible in the arteries is never heard except when aortic incompetence exists." With this opinion my own experience leads me to agree; there can be no doubt, moreover, that he is quite correct in his remark:—"aortic incompetence exists in many cases in which no such murmur is audible."

DIAGNOSIS.—The recognition of aortic incompetence is as a rule easy. The peculiar character of the radial pulse, the behaviour of the arteries in general, the condition of the left ventricle, and the diastolic murmur along the right edge of the sternum form a group of symptoms which are unmistakable. If to these there should be added capillary pulsation and a double murmur in the larger arteries, the diagnosis is by so much rendered the more secure.

The diagnosis of this affection is sometimes rendered less easy by individual peculiarities, chief among which must be mentioned the exceptional position of maximum intensity of the diastolic murmur to the left of the sternum, as in Case 25, p. 517, narrated below. This may lead to a possibility of error, but if there should happen to be excessive pulsation of the arteries, along with a capillary pulse, and no clubbing of the fingers, there will be little likelihood of such a mistake being committed. To this subject, however, further reference will be made in dealing with pulmonary valve disease.

The remote possibility of mistaking the diastolic murmur often found in mitral obstruction for that of aortic incompetence should not be overlooked, but the differentiation of the two murmurs and of the diseases in which they are found is as a rule perfectly easy. The diastolic mitral murmur is heard with its maximum intensity in the neighbourhood of the apex beat, and the character of the murmur is as a rule harsh. These two points in most cases suffice to distinguish

the murmurs, and it is hardly necessary to add that in uncomplicated mitral disease the arterial and capillary phenomena of aortic disease are absolutely wanting.

It is held by some authors that an aortic aneurysm, or a dilatation of the aorta, may, in the total absence of any implication of the aortic cusps, give rise to a double murmur. This cannot be accepted as a common occurrence. If a double murmur is found in any case, whether there is absolute or relative obstruction, the aortic cusps are incompetent. There can be no question of differential diagnosis in such conditions, and the whole clinical features are those of two distinct lesions present at the same time. The few exceptions which may be held to prove the rule will be referred to in the chapter on aneurysm.

The lesions in aortic incompetence may undergo considerable alteration, and a healing process resulting in recovery has been seen. In one most interesting case described by Walshe, incompetence of the aortic cusps passed into stenosis by a process of endocarditis. On the other hand, lesions in a case of traumatic aortic incompetence have been seen in which cicatrization had taken place. Both of these cases, however, must be regarded as pathological rarities.

PROGNOSIS.—Many diverse views have been held as to the gravity of aortic incompetence. It is generally recognised as being the cardiac lesion in which above all others the life of the patient hangs by a thread, but Stokes held stoutly to the contrary opinion that mitral disease tended to sudden death. The prognosis is dependent upon several different factors, such as the cause and nature of the aortic disease, the presence or absence of other valvular lesions, the integrity or implication of the arterial walls, the condition of the kidneys, and the general condition of the entire system with regard to nutrition. Speaking generally, those cases in which the disease has arisen from endocarditis have a more hopeful outlook than those belonging to the degenerative type, inasmuch as the latter are more likely to produce interference with the coronary circulation. Instances of aortic incompetence, uncomplicated by any other valvular lesion, afford a more hopeful prognosis than those in which there

is such a complication. Degenerative changes in the arteries, especially if attended by anginous seizures, necessarily lead to a less hopeful forecast, seeing that in such circumstances there is great liability to interference with the coronary circulation. If there be any interference with the circulation through the kidneys there will be much greater strain thrown upon the damaged heart; such a combination is thus of evil import. If the general nutrition of the individual, further, is at a high level, there is more hope of the heart surviving for a longer period the damage which it has sustained.

If hypertrophy keeps pace with the circulatory disturbance, if the second sound is distinct in the cervical arteries, and if the pulse pressure is good, the outlook is favourable; but if the cardiac systole is enfeebled, the sounds weakened, and the pressure of the pulse lowered, the future cannot be regarded otherwise than with apprehension.

TREATMENT.—Cases of aortic incompetence do not call for any special treatment beyond attention to such general rules as will necessarily be dictated by the presence of a lesion leading to considerable hypertrophy. The avoidance of fatigue, whether muscular or mental, careful attention to diet, and caution in the use of alcohol and tobacco, must be enjoined, while a healthy life with regard to food, exercise, and rest must be recommended.

Some special points nevertheless require a word in passing. In dealing with any case which is of sclerotic origin, with rigidity of the arteries and anginous attacks, the use of the iodine series of drugs is followed by marked benefit. Of all those drugs iodide of potassium is of the greatest importance, as has been recognised since it was first recommended by Graves. The dose does not require to be large, but the employment of the drug must be long continued. Doses of five grains three times a day may be continued for months without any result other than improvement of the symptoms. Sometimes iodide of potassium is, on account of some idiosyncrasy, badly tolerated, in which case iodide of sodium may take its place in similar doses to those just mentioned. The syrup of hydriodic acid may be employed, when neither of the salts is suitable, or iodine wine may be tried. For further



remarks on the treatment of anginous attacks, reference must be made to the section of this work dealing with that subject.

If a failure of compensation leads to a breakdown of the balance of the circulation, the treatment must be based upon the principles which have been laid down in the general remarks on treatment. The only point to which reference need be made at present is in regard to the long controversy over the employment of digitalis. In Corrigan's original memoir he discountenanced the use of digitalis in aortic disease, and in this he has been followed by many writers, especially by Fothergill, and, to some extent, by Morison. The fears which have been expressed are based upon the supposed evil of prolonging the diastolic period, and so allowing the regurgitation to exert its baneful influence during a greater length of time on the left ventricle. These ideas, however, are scarcely to be regarded as substantial, and everyday experience teaches that Balfour is right in his contention that digitalis or one of its congeners is imperatively demanded when cardiac failure makes its appearance in aortic incompetence. It must be remembered that, as Broadbent has well put it, there are aortic cases with mitral symptoms. These must be treated like mitral cases.

CASE 19. *Aortic Incompetence*.—E. O'N., aged 38, married, miner, presented himself in the medical waiting-room of the Royal Infirmary, 17th October 1892, complaining of pain in the chest.

The patient was unable to give any very distinct account of his family history. His own health had always been good until the present illness began, and he had particularly never suffered from rheumatism in any form. His occupation had involved a good deal of hard work, but no excessive strain. For some weeks he had experienced some vague uneasiness, often amounting to pain, in the chest; he was, however, unable to localise it distinctly. He complained of no breathlessness.

On examination the alimentary and blood-glandular systems furnished no evidence of disturbance. The patient's skin was somewhat pale, and on reddening a portion of the forehead a distinct capillary pulse was to be seen. It was difficult to say whether there was anything of the kind in the finger nails. All the superficial arteries throbbed violently. The radial artery was somewhat hard, but not tortuous. Between the pulsations it gave a sensation of being empty, but the pressure was fair; its rate was between 60 and 70, and its rhythm perfectly regular. Each individual pulsation was swift and large, collapsing, however, at once, and giving, therefore, the well-marked characters of Corrigan's pulse. The apex beat

was in the sixth intercostal space, four and three-quarter inches from mid-sternum. No thrill could be made out on palpation. The area of cardiac dulness extended three inches to the right and five inches to the left of mid-sternum. On auscultation the first sound was perfectly clear over the whole præcordia, and there was no trace of a murmur in the carotid arteries. The second sound was replaced by a loud and somewhat high-pitched diastolic murmur heard over a large area, but having its maximum intensity about half-way down the sternum.

There were no symptoms of disorder connected with any other system of the body.

The patient in this case was one of the most remarkable instances of uncomplicated incompetence of the aortic cusps ever seen by me, and the causation was probably some sclerotic change producing shrinking of one or more of the aortic cusps.

*CASE 20. Aortic Obstruction and Incompetence.*—J. R., aged 28, a married woman engaged in gardening, was admitted to Ward 25 in June 1891, suffering from cough and pain in the side. Her father died at the age of 50 of heart disease. Her mother was in good health. She had one brother and three sisters all well, no member of the family having died. Her social conditions had always been indifferent. Except for an attack of typhus fever when young, the patient's health had always been good until a few years before admission, when she had been troubled with some breathlessness. The patient had been suffering from pain in the chest for about three weeks before admission, and as this gradually became worse she presented herself in the waiting-room.

On admission the patient was found to have a temperature of 103·5°; the pulse rate was 108, that of the respiration 42. There was a deep flush upon both cheeks, and there was profuse perspiration. A considerable amount of diarrhœa was present on admission and for a few days thereafter. The tongue was moist and covered with a thick fur. No other digestive symptoms were present. The spleen was not enlarged and the lymphatic glands were in no respect altered. On examination of the blood a considerable degree of leucocytosis was found, as will be seen from the following table, kindly furnished by Dr. A. C. E. Gray, the resident physician :—

	Red Corpuscles.	Ratio of White to Red.
17th June	5,500,000	1 to 100
18th    "	5,500,000	1    "   75
19th    "	5,750,000	1    "   55
20th    "	5,675,000	1    "   56
21st    "	5,350,000	1    "   78
22nd    "	5,875,000	1    "   75
23rd    "	5,700,000	1    "   80
24th    "	5,500,000	1    "  110
25th    "	5,512,500	1    "  110

From the 25th onwards the ratio rapidly came back to the normal.

On examination of the pulse the wall of the artery was found to be soft and yielding. The vessel was moderately full and the pressure was about the normal. The pulsation was bounding and collapsing, presenting, in short, the typical characters of the water-hammer pulse. A tracing of it, obtained with the sphygmograph, is given in Fig. 143. On examining the neck the only appearance observable was excessive pulsation of the carotid arteries. There was no morbid appearance connected with the cervical veins. On the back of the hand a direct venous pulse could be seen travelling upwards from

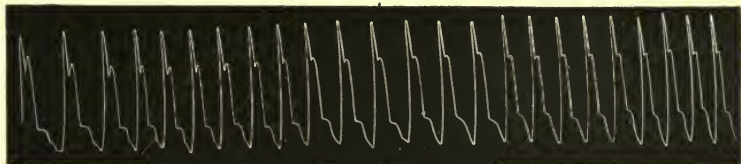


FIG. 143.—Tracing from acute pneumonia in a patient suffering from aortic regurgitation ; pressure 2 oz.

the knuckles to the wrist. This followed the systole of the heart by a considerable interval. A tracing obtained from it by means of an extremely light lever is seen in Fig. 144. On causing a red patch on the forehead by friction, a very distinct capillary pulse could be seen. Inspection of the præcordia did not reveal any abnormality, save the fact that the apex beat situated in the fifth intercostal space was farther to the left than in health. There was no fremitus on palpation. The borders of the heart as ascertained by percussion were one inch and



FIG. 144.—Tracing from vein on the back of the hand in a patient with acute pneumonia and aortic incompetence.

a half to the right, and four and three quarter inches to the left, of mid-sternum. The superior border of the heart was at the upper level of the third rib. On auscultation two distinct murmurs were determined, one of which, systolic in time, had its maximum intensity at the right edge of the sternum opposite the first intercostal space. It was loud and rough, and was distinctly propagated up the arteries of the neck, and along the subclavian arteries as well. The diastolic murmur had its maximum intensity about mid-sternum opposite the fourth costal cartilages, but it could be heard distinctly at the apex of the heart, as well as at the xiphoid cartilage. The patient suffered from a considerable amount of cough attended by a characteristic rusty viscid sputum. On examination of the chest it was found that the right side did not move so freely as the left, that there was greatly increased vocal fremitus over the lower lobe,

which was dull on percussion, and that over the whole of the lower lobe there was high-pitched bronchial breathing, with a few fine crepitations towards the end of inspiration, and greatly exaggerated vocal resonance. Over the whole of the left lung the breathing was puerile, but there was no other departure from healthy conditions. The urinary secretion was slightly diminished, of high specific gravity, and dark colour. It contained a considerable increase of urates, and almost no chlorides. There were no abnormal phenomena connected with the nervous system.

The patient was treated by means of copious nourishment, free alcoholic stimulation, and a combination of digitalis and ammonia, under which she made a rapid and complete recovery from the pulmonary condition, and left the hospital in due course.

It was obvious that the patient in this case was suffering from acute lobar pneumonia in its most characteristic form. It is probable that the pyrexia which accompanied it was in part at least responsible for the direct venous pulsation which formed one of the chief points of interest in the case.

Properly speaking this case should be placed amongst instances of combined obstruction and regurgitation, but, seeing that they have been described to illustrate the direct venous pulse of aortic incompetence, they may well have a place here.

## AORTIC OBSTRUCTION AND INCOMPETENCE.

As has been already insisted upon, aortic obstruction and aortic regurgitation are in themselves much less common than a combination of the two lesions. The Royal Infirmary statistics, previously given in detail, show that out of 411 cases of aortic disease, 141 were diagnosed as aortic incompetence, 40 as aortic obstruction, and 230 as combined obstruction and incompetence. These figures mean that considerably more than half of all the aortic cases have the combined lesions. These figures do not, however, appear to me to be even approximately correct. Aortic incompetence in itself is rather a rare affection, and that it should occur two and a half times as commonly as aortic obstruction, is to me matter for surprise. There can be no doubt that many cases of double aortic disease are returned as aortic incompetence, simply because that aspect is the most outstanding feature.



ETIOLOGY AND MORBID ANATOMY.—But little requires to be said on these heads. The factors which produce the double lesions are the same as those concerned in the origin of either obstruction or regurgitation. As regards the structural alterations which are present, they show similar changes to those which have been already described; but in every case where the twofold effects of obstruction and regurgitation are present, there is always some roughening or thickening of the cusps, along with some retraction or deformation which hinders their closure.

SYMPTOMS AND DIAGNOSIS.—The essential features produced by combined obstruction and incompetence are the mingling of the appearances peculiar to each. The clinical picture is, therefore, somewhat more shifting than is the case as regards either lesion in itself. While the general symptoms cannot be held to depart from those manifested by either lesion, the effects are usually more severe, and the symptoms, therefore, are apt to be more pronounced. Physical examination of the circulatory organs reveals the characteristic features belonging to each of the two lesions which are combined. Sometimes the one set, and sometimes the other, are in the ascendant. The pulse while perfectly regular and equal, as it is in either lesion, sometimes gives the characteristics of the *pulsus tardus*, at other times of the *pulsus celer*. The apex beat is, as might be expected, displaced downwards and outwards, and thrills systolic, or diastolic, or both, may be present. The area of cardiac dulness is considerably increased, and systolic and diastolic murmurs are heard.

From the point of view of diagnosis, nothing need be added here. The determination of either lesion must be conducted on the principles already laid down.

PROGNOSIS AND TREATMENT.—While the combined lesions produce a state of matters much more serious than obtains in the case of a simple aortic obstruction, it cannot be held that the combination is much more serious than simple aortic incompetence. This belief must be allowed to have no absolute foundation on statistical evidence. It, nevertheless, is the result of much careful observation.

The treatment of combined aortic lesions comes practically

to be that of aortic incompetence, seeing that it constitutes the most disturbing element in the combination. Nothing, therefore, need be added to what has been already said upon this subject.

CASE 21. *Aortic Obstruction and Regurgitation.*—T. S., æt. 65, horse-dealer, has frequently consulted me in the out-patient department of the Royal Infirmary on account of weakness, shakiness, palpitation, and uneasiness in the chest.

His father died at the age of 77 from the effects, on the patient's showing, of alcohol; his mother at the age of 94, of old age, according to him. He had three brothers, all of whom died some years before the patient was first seen by me, one from an accident, and the two others in consequence of acute pulmonary affections induced by irregular habits and imprudent exposure. Three sisters, alive at the date of my last seeing the patient, had always enjoyed excellent health. The patient's social conditions might have been eminently satisfactory for his position in life but for his dissipated habits. According to his own statement he had been greatly addicted to alcohol, and his general conduct had been sadly in need of reformation. His previous health had always been satisfactory, and the onset of the malady for which he sought advice had been in the highest degree insidious.

The patient had a high complexion, and venous stigmata were dotted abundantly upon the cheeks and nose; the tongue was very tremulous

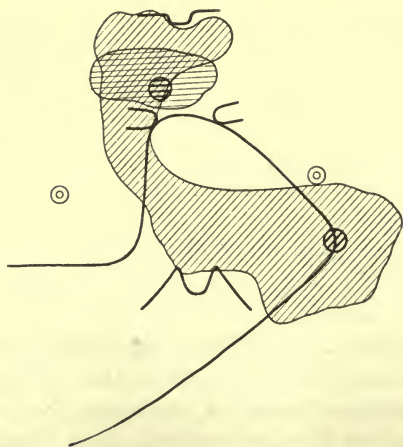


FIG. 145.—Aortic systolic and diastolic murmurs.

and somewhat furred; the digestive functions otherwise were satisfactory. There was great dilatation of the right pupil. The arteries of the neck throbbed very distinctly, and there was also great pulsation of the right subclavian artery. The radial artery was hard and tortuous, as were the other superficial arteries throughout the body. The pulse wave was large and bounding, but well sustained; its rate was usually about 70 and it was perfectly regular. On palpation of the arteries of the neck, a systolic thrill

was perceptible, and this was more distinct in the right subclavian artery than elsewhere. The apex beat was in the sixth intercostal space, five and a half inches from mid-sternum; it was unaccompanied by any

thrill—none, indeed, could be felt over any part of the præcordia. On percussion, the right border of the heart at the level of the fourth costal cartilage was two inches from mid-sternum; the left border at that level was three and a half inches from mid-sternum, and in the fifth and sixth intercostal spaces it was five and a half inches from mid-sternum. On auscultation two distinct murmurs were present. A systolic murmur with its maximum intensity over the manubrium sterni was heard at the junction of the second rib with its right border. Its area of audibility was a small one, being confined to an oval space surrounding the maximum intensity and measuring five inches in length by two and a half inches in breadth. This murmur was somewhat harsh in its character. A diastolic murmur was also heard with its maximum intensity at the apex. The area over which it could be heard showed a most curious distribution; mapped out on the surface of the chest, it extended from the upper border of the manubrium sterni to somewhat below and outside of the apex beat, forming a figure somewhat resembling a boomerang in shape. This murmur was, like the systolic one, somewhat harsh in its character.

There were no respiratory or urinary symptoms, and with regard to the nervous system there was no appearance of any disturbance apart from considerable tremulousness of the whole muscular system, and the dilatation of the right pupil already referred to.

In this case it was perfectly clear that the patient presented combined obstruction and incompetence of the aortic valve, produced apparently by long-continued physical stress and alcoholic abuse. The points of greatest interest in the case were the curious distribution of the cardiac murmurs and the fact of some dilatation of the subclavian artery, producing irritation of the right sympathetic nerve.

CASE 22. *Aortic Obstruction and Regurgitation*.—M. A., aged 43, married, housewife, was admitted to Ward 25 of the Royal Infirmary on 16th May 1892, complaining of breathlessness and swelling of the ankles. Her father fell victim, at the age of 45, to cardiac disease resulting from rheumatism. Her mother died, when 80, of chronic bronchial troubles. She had never had any brothers or sisters, but had two daughters, both in excellent health. Her surroundings had always been good, and her health excellent; more particularly, she had never suffered from rheumatism. Four or five weeks before admission she observed some swelling of the ankles, and began to suffer from breathlessness.

On admission, there were no special symptoms of alimentary or hæmopoietic origin. The patient was pale. The subcutaneous tissues were very œdematous in the dependent parts. No capillary pulse could be observed anywhere. The superficial arteries were somewhat rigid and slightly tortuous. The radial pulse had more than the normal pressure. Its rate was 100. It was perfectly regular, and the pulsations were small and sustained. There was no excessive throbbing in the vessels of the

neck. The apex beat was in the sixth intercostal space, three and three-quarter inches from mid-sternum. No thrill was observable. The cardiac dulness extended two and a half inches to the right, and four inches to the left, of the middle line. On auscultation at the base of the heart, two murmurs were heard, corresponding to the systole and diastole. They were both somewhat harsh in character. The former had its maximum intensity over the manubrium sterni and was propagated into the vessels of the neck. The latter had its point of maximum intensity opposite the third costal cartilage, and was extended to the xiphisternum. In the mitral area there was a systolic murmur, harsh also in its character, but different in tone from the systolic murmur at the base. It was conducted into the axilla and round to the angle of the scapula. In the tricuspid area there was yet another murmur, softer altogether in its character than the two systolic murmurs which have been described.

On examination of the respiratory system, there was some muffling of the percussion sounds at the bases of both lungs, and there were abundant moist crepitations. The patient was unable to sleep in the recumbent posture on account of the severe dyspnœa. The urinary secretion was scanty and of high specific gravity, containing a small quantity of albumin.

In this case it was obvious that a double aortic lesion was present, probably of sclerotic origin, and it seemed probable from the character of the mitral systolic murmur that there was also an organic mitral lesion, together with dilatation of the right ventricle. In spite of sedulous attention, the patient steadily grew worse, and notwithstanding stimulating treatment of every kind she died.

The following is an abstract of the autopsy, performed by Dr. Muir :—

*External Appearances.*—Rigor and lividity were present, the face and neck being livid. Considerable œdema. Striæ albæ were seen on the abdomen and thighs.

*Thorax.*—Rather more than two pints of fluid were found in the right pleura. There were pleural adhesions over the upper half of the left lung, while, in the lower half of the cavity, there were 12 oz. of fluid, and the adhesions also were œdematous. There was about 1 oz. of fluid in the pericardium.

*Heart.*—Its weight was 1 lb. 2 oz. There was a large milk spot on the right ventricle, and a similar one on the left. There was recent soft black clot in both ventricles. The aortic valves were incompetent. The cone diameter of the aortic orifice was 0·3". The valves were united, rigid, and calcareous. The pulmonary diameter was 1·0". The mitral orifice was also contracted, the valves being thickened, fibroid, and calcareous. Its cone diameter was '55". The tricuspid orifice measured



1.25". The left ventricle was hypertrophied and fibroid; its wall was firm. The right ventricle was dilated, and its wall was hypertrophied. Both auricles were dilated and hypertrophied.

*Lungs.*—The left lung weighed 1 lb. 10 oz. It was congested and œdematous, and the pulmonary artery was atheromatous. The right lung weighed 1 lb. 6 oz., and resembled the left.

The chief interest here lay in the very small aortic orifice shown in Fig. 138, p. 475.

CASE 23. *Aortic Obstruction and Regurgitation.*—J. C., aged 31, a married tailor, formerly a trooper in the Scots Greys, was admitted to Ward 22 of the Royal Infirmary, in August 1893, on account of palpitation. His father and mother were both alive and in good health. His wife had never had any children. His social conditions had always been satisfactory. In November 1891, the patient suffered from acute rheumatism, and had never been well since that date. The alimentary system presented no features of interest, and there was no alteration in the blood-glandular system. The pulse was, as a rule, about 80, and presented the well-marked features of Corrigan's pulsation. There was a well-marked capillary pulse in any area that had been reddened. The carotid arteries pulsated in a very exaggerated manner. On inspection of the præcordia a tremendous heaving could be seen, not merely excessive in amount of movement, but also in its extent. The apex beat could be seen in the sixth intercostal space, five and a half inches from mid-sternum. On palpation the force of the impulse was found to be enormous, but there was no thrill. The right border of the heart was three inches, and the left border five inches from mid-sternum at the fourth costal cartilage, but at the upper level of the xiphoid cartilage it was six inches from mid-sternum. Measured diagonally from the right border at the fourth costal cartilage to the left border at the xiphoid level, the heart measured nine and a quarter inches. The upper border was at the level of the lower part of the second intercostal space. On auscultation a loud rough systolic murmur was heard most distinctly over the right half of the manubrium sterni about half-way up, and there was also a diastolic murmur somewhat softer in character, but also extremely loud, whose point of maximum intensity was in mid-sternum about the level of the fifth costal cartilage. The systolic murmur was propagated over a considerable distance along all the arteries, and throughout most of the chest. The diastolic murmur had also a somewhat wide distribution.

In this case a very large amount of dilatation had accompanied the development of the hypertrophy. As a contrast to it the following is of interest:—

CASE 24. *Aortic Obstruction and Regurgitation.*—P. R., aged 38, a married labourer, was admitted to Ward 22, in August 1893, complaining of pain in the chest. His father was 94 years old, and in remarkably good health, considering his age. His mother

had died when 70 years old of chronic bronchial trouble. In early life the patient had acquired specific infection, and he had suffered from acute rheumatism nine years before the date of his admission. He had about six years previously developed a popliteal aneurysm, for which he had been treated in London by Mr. Rickman Godlee. Six weeks before his admission he began to suffer from the pain in the chest, and this became so severe that he found it necessary to seek advice. There were no indications of disturbance connected with any system excepting the circulatory. The pulse varied between 75 and 85, the artery was resistant and slightly tortuous, the vessel was moderately full, the pressure was rather high, the pulsation was bounding and collapsing, manifesting in brief most features of the water-hammer pulse. A distinct capillary pulse could be seen over a reddened area on the forehead. All the visible arteries pulsated very freely. On examination of the præcordia, the apex beat was seen to occupy the fourth left intercostal space, three and a half inches from mid-sternum. No thrill could be ascertained on palpation. The right border of the heart was one inch and three-quarters to the right, and the left was four inches to the left, of mid-sternum at the level of the fourth costal cartilage. The upper border of the heart was in the second left intercostal space at its lower part. Systolic and diastolic murmurs were present, the former with its point of maximum intensity half-way up the manubrium sterni and a little to the left of the middle line, propagated in the carotid and subclavian vessels, as well as throughout a considerable part of the chest; the latter heard best at mid-sternum half-way between the levels of the third and fourth costal cartilages, but distinctly carried to the xiphisternum and the apex.

In this case it seemed probable, from the existence of the anginous symptoms, that the small size of the heart was conditioned by some interference with the coronary circulation, very likely of the nature of sclerosis.

As showing that aortic murmurs may have their loudest intensity about the pulmonary area, the following case is useful:—

CASE 25. *Aortic and Mitral Disease.*—Esther C., æt. 46, housewife, was admitted to Ward 25 of the Royal Infirmary on 3rd July 1893, suffering from dropsy and dyspnœa.

The patient was so ill that it was not possible to disturb her by any prolonged examination, but it was ascertained that she had suffered more than once from acute rheumatism, and that four years before admission she had been attacked by hemiplegia of the right side.

She was deeply cyanosed, and could only breathe when sitting upright in bed. The fingers and toes were most distinctly clubbed, and there was much arching of the nails. The right side of the face was rather vacant in expression, and its muscles were weak, while the right arm and leg were almost powerless. The feet, legs, and thighs were very œdematous,

and there was some ascites. The pulse was irregular, and did not give any definite indications, although the pulse wave was of brief duration. The cardiac impulse was feeble, but the apex beat was felt in the sixth intercostal space. No thrill could be discovered. On account of the patient's condition, it was not considered right to trouble her by percussing the præcordia, and the size of the heart was not therefore ascertained. For the same reason it was not possible to study the auscultatory phenomena so fully as could have been wished, but the following facts were definitely established. At the apex of the heart there was a rough first sound, accompanied by a blowing systolic murmur propagated into the axilla, and also, but to a less degree, towards the sternum. At the base, a rough systolic was followed by a softer, although still somewhat harsh, diastolic murmur. Both of these murmurs were heard over the whole of the upper part of the chest, but their maximum intensity was to the left of the sternum, in the neighbourhood of the third costal cartilage. The diastolic murmur, in particular, had its point of maximum loudness in the third left intercostal space, about an inch to the left of the sternal edge. The systolic murmur was distinctly propagated along the carotid arteries. The respiratory system showed symptoms and physical signs of hydrothorax and œdema of the lungs. The urine was scanty, and contained albumin. The patient had almost complete paralysis of the right leg and arm, with weakness of the facial muscles of the right side, and on the same side there was a considerable degree of wasting of the muscles, with contracture.

In this case the original clinical features were to a great extent modified by the obvious failure of the cardiac muscle. It could not be doubted that there was a double lesion at the mitral orifice, producing obstruction and incompetence. It was clear also that there was aortic obstruction and incompetence. But, as has been noted, the maximum intensity of the diastolic, as also of the systolic murmur, was to the left of the left sternal border, which fact, taken along with the clubbing of the fingers and toes, together with the profound respiratory disturbance, caused some doubt whether there might not be some lesion of the pulmonary valve or orifice. The conclusion arrived at, however, was that, in consequence of the backward pressure resulting from the mitral lesion, the clubbing of the fingers had resulted, while the combined effect of the mitral and aortic affections had been to cause the serious condition of cardiac failure.

In spite of all treatment the state of the patient became gradually worse, and she died somewhat suddenly on the 12th July. The post-mortem examination was performed on the 14th July by Dr. Muir, whose notes are appended.

*External Appearances.*—Body well nourished. Some dropsy of lower limbs. Rigidity well marked.

*Thorax.*—Left pleura contained 24 oz. of serous fluid, with fibrous adhesions here and there; right pleura, about 15 oz. Pericardium contained about 3 oz. serum.

*Heart* considerably enlarged on both sides. The right auricle was



somewhat dilated, and filled with dark clot. Aortic orifice distinctly incompetent. Pulmonary orifice competent.

The cone-diameters were as follows:—aortic = .65 ; pulmonary = 1 ; mitral = .7 ; tricuspid = 1.7. The aortic cusps showed extensive chronic vegetative endocarditis. (a) The two posterior cusps were much thickened at their junction and also calcareous. The margins were also thickened. (b) The anterior cusp was considerably ulcerated, and there were large irregular vegetations hanging down from it. The orifice was stenosed from the chronic endocarditis, and also markedly incompetent. The mitral segments were much thickened and indented, and had become adherent for a considerable distance, giving rise to stenosis. The chordæ tendineæ were also thickened and contracted somewhat, as were also the apices of the papillary muscles. There was also evidence of more recent endocarditis along the margins of the segments. The tricuspid and pulmonary valves were normal.

Left ventricle, 4 in. in length by  $\frac{5}{8}$  to  $\frac{3}{4}$  in. in thickness ; slightly dilated and hypertrophied. Right ventricle much hypertrophied ; its walls measured  $\frac{5}{16}$  in. in thickness. The inner surface of both ventricles showed commencing fatty change in the myocardium. No ante-mortem thrombi.

*Lungs.*—Right, 1 lb. 12 oz. ; showed chronic venous congestion. The bronchi were deeply congested, and contained frothy secretion. The posterior part of the lower lobe was going on to hypostatic pneumonia. There was also slight interstitial change in places. Left, 1 lb. 8 oz. ; at the posterior part about middle line there was anasarca, in which part the bronchi were considerably dilated, with considerable interstitial change around. Some of the bronchi contained a purulent fluid. There was a similar patch in the lower lobe. The general condition resembled that of the other lung.

*Abdomen.*—Peritoneum contained about 40 oz. of serous fluid.

*Liver.*—Weight, 2 lb. 10 oz. ; it showed chronic venous congestion. The tissue was indurated, and the capsule in places was thickened.

*Spleen.*—Weight, 10 oz. ; showed typical chronic venous congestion.

*Kidneys.*—Each weighed 7 oz. ; they showed foetal lobulation, and also several irregular depressions owing to old infarcts. The infarcts had been in both kidneys, but were more numerous in the left.

The other organs showed chronic venous congestion. There were no recent infarcts.

*Brain.*—There was nothing abnormal to see on the surface, and the vessels at the base were practically normal ; none appeared to have been the site of embolism. On opening the brain, there was found an old area of softening in the left side, which implicated the following structures:—at its lowest level, the posterior part of the lenticular nucleus, the external capsule and the claustrum, with the convolution (in part) of the island of Reil, were softened. The posterior end of the internal capsule was also affected, and there were one or two minute points of softening in the optic thalamus. Higher up, the lenticular nucleus was more destroyed, but the internal capsule was practically unaffected. The softening ran up for some distance along its outer aspect.



The convolutions (with the exceptions mentioned) were unaffected. No change could be seen on naked-eye examination either in the pons or medulla.

The morbid anatomy of the heart is shown in Fig. 137, p. 474.

The consideration of the clinical and pathological facts presented by the patient whose case has been described, leads to the conclusion that the mere position of the maximum intensity of murmurs in itself may lead to erroneous conclusions; and that even when definite appearances, strongly suggestive of pulmonary lesions, are present, they may be susceptible of other explanations.

That the murmurs produced at the aortic orifice may be loudest in, or even to the left of, the pulmonary area, is a well-known fact. As one of several patients presenting this feature, which have come under my notice, the clinical features of the following case may be shortly described:—

CASE 26. *Aortic Obstruction and Incompetence*.—Bella W., æt. 19, domestic servant, presented herself as an out-patient at the Royal Infirmary on 29th May 1893, complaining of a rash on her skin.

Her family history was in every respect satisfactory, and her sur-

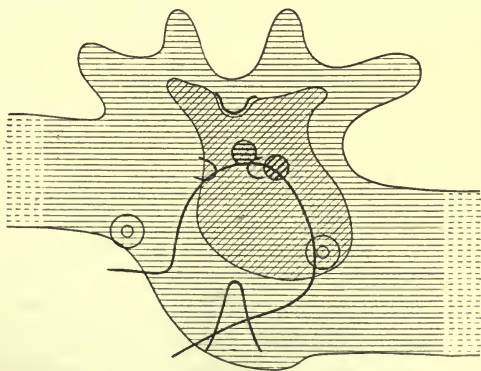


FIG. 146.—Aortic systolic and diastolic murmurs.

roundings had always been favourable. She had suffered from acute rheumatism some years ago, but had otherwise enjoyed good health. The affection of the skin—well-marked psoriasis—had for some months been gradually increasing; with the exception of slight uneasiness in the præcordia, it was the only symptom of which she complained.

The alimentary and hæmopoietic systems showed no abnormal phenomena. The pulse was usually about 80 per minute, and perfectly regular; the vessel was somewhat empty, and the tension low; the pulse wave was large and bounding, but short and collapsing,—in a word, it was a well-marked water-hammer pulse. The arteries of the neck pulsated rather strongly. Inspection of the præcordia showed no abnormality, and, on palpation, the apex beat could be felt beating strongly in the fifth intercostal space. No thrill was present. The deep cardiac dulness extended from  $2\frac{1}{2}$  in. to the right to  $2\frac{1}{2}$  in. to the left of the mid-sternal line. On auscultation, two very distinct murmurs were heard, systolic and diastolic in time. The systolic murmur was loudest at the junction of the second left costal cartilage with the sternum, and was propagated up the neck, out to the shoulders, and around the thorax on both sides to the vertebral column behind. The diastolic murmur had its maximum intensity in the second left intercostal space rather more than  $1\frac{1}{2}$  in. to the left of the sternal border, and it was conducted to the upper end of the manubrium sterni, along the clavicles, and over most of the præcordia. The distribution of these murmurs is shown in Fig. 146.

There was no clubbing of the fingers, and no symptoms of backward pressure were present; in fact, no other point of clinical importance, except the psoriasis, was found.

This case shows very well how, in an undoubted instance of aortic disease, the auscultatory phenomena were most distinct in the pulmonary area. Such instances might be multiplied almost indefinitely, were it necessary to do so.

## CHAPTER XI.

### AFFECTIONS OF THE MITRAL ORIFICE.

THERE is an essential difference between the two disorders which may be present at the mitral orifice. Mitral obstruction is comparatively rare as an isolated lesion. In the preponderating majority of instances it is accompanied by incompetence of the valve. It is far otherwise with regard to mitral incompetence. Mitral incompetence, it will be seen, may be produced by causes and lesions exerting no influence upon the free access of blood from the auricle into the ventricle; it therefore often exists without any obstruction. It must, nevertheless, be admitted that mitral incompetence as an isolated lesion is far from common. As the result of endocarditis it is usually an attendant upon obstruction. Frequently the determination of the latter lesion is difficult, and the diagnosis is more a matter of inference than demonstration, yet from the results of clinical observation and pathological investigation there can be no doubt of the usual combination of the two lesions. When, on the other hand, mitral incompetence occurs from causes other than lesions of the cusps, it is in the overwhelming proportion of cases associated with incompetence also of the tricuspid valve. Facts in support of this view will be adduced in the proper place.

### MITRAL OBSTRUCTION.

Obstruction of the mitral orifice was recognised as a lesion in the early period of the modern study of medicine, but its

recognition from the clinical point of view has been gradually developed during the present century. Morgagni and other authors very fully described lesions found in this affection. Corvisart showed the value of the thrill which is so frequently found in mitral obstruction, and Fauvel discovered the distinctive murmur which accompanies this disease. The further development of our knowledge of the affection is due to so large a number of industrious workers that it seems almost invidious to cite any as more particularly worthy of mention, yet on account of the value of their observations, the writings of Stokes, Gairdner, Hayden, Balfour, and Hilton Fagge must be particularly mentioned.

The presystolic murmur of mitral obstruction appears to have been heard by Bertin, who, as Hayden says, "actually founded thereon the positive diagnosis of left auriculo-ventricular contraction." As he, however, believed with Laennec that the second sound was produced by the contraction of the auricles, he regarded the murmur as being diastolic in rhythm. Adams, three years later, and apparently quite independently of Bertin's observations, made out some of its special characters, but apparently did not grasp the exact rhythm of the murmur or of the attendant thrill. Valuable though the writings of Hope are, there is in his first edition a total misconception of the phenomena of mitral disease, and even up to the third edition of his work this subject is left in considerable obscurity. The real clinical history of mitral obstruction begins therefore with Fauvel.

ETIOLOGY.—Obstruction at the mitral orifice is more common in women than in men. The relative proportion in which the two sexes are affected necessarily fluctuates somewhat in different statistics, but the facts of the admissions to the Royal Infirmary are that for 133 males admitted in five years there were 171 females.

The affection is one essentially of early life, for although often not detected until a later period, it is found, on diligent inquiry, to have had its beginnings at a much earlier period. According to the Edinburgh statistics the largest number of the admissions occur between the ages of twenty and thirty.

The disease is sometimes even congenital, although patients



do not come under treatment for it during the first decade of life to any considerable extent. According to some observers mitral obstruction is to be regarded as a developmental lesion.

Mitral obstruction rests more frequently upon endocarditis as a causative factor than does aortic obstruction. Statistics bearing upon this point vary within wide limits. The endocarditis may have its origin in any of the causes which have been previously considered, but rheumatism in one or other of its protean manifestations is beyond all question the most potent agent in its production.

In a certain proportion of patients the most diligent inquiry altogether fails to reveal any definite cause for mitral obstruction, inasmuch as there has been no history of any endocarditis, or of any of the affections which usually lead to that condition. It must, nevertheless, be allowed that in a certain proportion of instances there may be a latent cause operating in the direction of endocarditis, and it occasionally happens that years after a patient has suffered from mitral obstruction the development of certain aberrant forms of rheumatism throws a significant light upon the etiology of the cardiac lesion. Notwithstanding this admission, it must be granted that in some cases of mitral obstruction the causation is quite obscure.

It has been suggested by Potain that mitral obstruction may possibly prove to have its origin sometimes in pulmonary tuberculosis. He suggests that the cardiac lesion may be secondary to the tubercular infection and be produced by the bacilli in the circulation affecting the borders of the valves. Potain has observed that in fifty-five cases of pure mitral obstruction there were nine instances of pulmonary tuberculosis presenting fibrous or cretaceous lesions. It has already been shown that tubercular endocarditis has a real existence, and it is somewhat difficult to understand why, if there be any basis for the suggestion of Potain, there should only be a simple mitral lesion showing no tendency to tubercular endocarditis. The view of Potain is certainly one of much interest. If it should prove to have any reality it would be a beautiful example of a compensatory process, since there cannot be the

shadow of a doubt that mitral stenosis stands in direct antagonism to phthisis pulmonalis.

**MORBID ANATOMY.**—The lesions which produce mitral obstruction are found to occupy two distinct zones. In one group they are situated at the level of the orifice itself, *i.e.* at the level of the auriculo-ventricular ring. In the other they are connected with the cusps. Of these two positions of the lesions the latter is by far the more common.

At the level of the auriculo-ventricular ring there may be different types of structural alteration. Vegetations resulting from endocarditis form one well-marked group of lesions, while sclerotic processes, mostly of degenerative origin, constitute another. In old-standing cases, however, of mitral disease it is not an unusual occurrence to find a combination of both, in which, moreover, a certain amount of calcification has occurred.

Much more common than such lesions are changes undergone by the valves and the chordæ tendineæ. The lesions in this case are threefold in their variety. The cusps may be united by their margins to a greater or smaller extent, as Bouillaud was the first to emphasise and explain. The resulting lesion in this case is that a restricted aperture is left, varying very considerably in its size and form. Sometimes, apparently in consequence of considerable retraction of the cusps, the mitral orifice appears almost closed by a diaphragm perforated by the small aperture which is left. Such a lesion is shown in Fig. 166, p. 597. It is, however, much more common to find that the united cusps project downwards into the ventricle in a funnel shape. In many cases the aperture is termed a "button-hole" orifice in consequence of its close resemblance in form to that object. It is well shown in the illustration mentioned.

In such cases the chordæ tendineæ become almost invariably thickened and rigid, so that they must still further interfere with the movements of the cusps.

In another large group of cases the structural alterations are vegetative in type and consist essentially in an outgrowth of granulations resulting in verrucose structure. The customary position for such vegetations is the auricular surface of the cusps near, but not quite at the free border, as has already

been seen in dealing with endocarditis. These vegetations are often associated with thickening of, and deposits upon, the

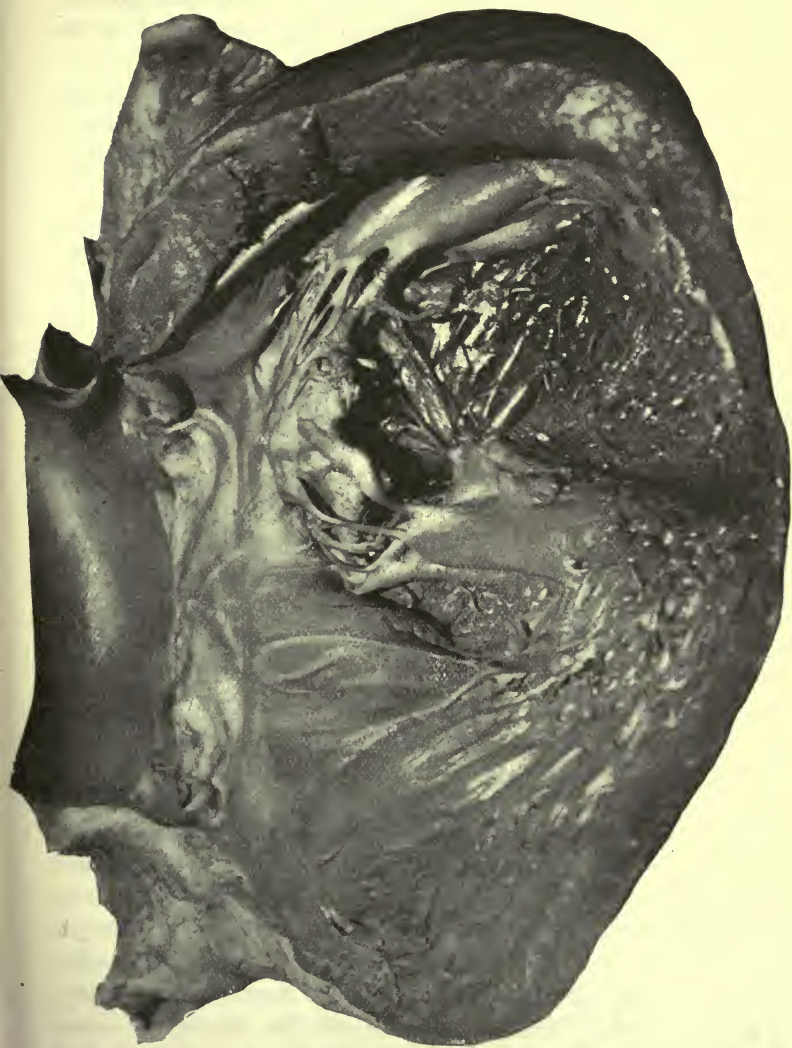


FIG. 147.—Mitral obstruction of vegetative type.

chordæ tendineæ, so that the cusps and their attachments present a greatly increased thickness with roughness and rigidity, as is shown in Fig. 147.



There are also changes produced apparently by purely sclerotic processes, consisting in thickening of the cusps with patches of deposit, frequently atheromatous and often calcareous. These result in deformities of the cusps, with great rigidity and interference with their proper functions.

Whatever be the nature of the process producing mitral obstruction, there are always certain well-marked consequences which follow in more or less direct ratio to the obstacle which is thereby present.

The effects of the interference with the forward passage of the blood are felt mostly by the left auricle. This was pointed out long ago by Adams. The consequence, as might naturally be expected, is a degree of hypertrophy proportional to the increased amount of work which the auricle has to undertake. A statement, repeated in many works, is that the result of mitral obstruction is, in so far as the left auricle is considered, a condition of dilatation and hypertrophy. This statement rests upon a somewhat unsubstantial foundation. It is perfectly true that in many old-standing cases of mitral obstruction, in which cardiac failure has occurred, there is dilatation along with hypertrophy, but, in cases uncomplicated by cardiac failure, hypertrophy without dilatation is undoubtedly the condition present. The recent observations of Samways have thrown much light on this subject.

Samways points out that during the four years 1888-91 inclusive, there were 70 cases of mitral obstruction reported on in the post-mortem room of Guy's Hospital. Of these 70 cases, 36 had severe obstruction, and 34 a less degree. Hypertrophy of the left auricle occurred in 36 of these cases. In 15 of the 36 severe cases the left auricle was hypertrophied without dilatation. In 11 other cases the auricle was both dilated and hypertrophied. In only 3 cases was the auricle dilated without being hypertrophied. No definite statements were made in regard to 7 cases. There was therefore hypertrophy of the left auricle in 26 of the 36 severe cases. Dilatation existed only in 14 cases, and was associated with hypertrophy in 11 of these cases. That dilatation is chiefly a breakdown phenomenon, Samways concludes from the following consideration. The 70 cases referred to included 18



which came from the surgical wards. In only one of these cases was there dilatation of the auricle. The absence of dilatation in the surgical cases, and its absence in the majority of the slighter medical cases, leaves very little basis for the assumption that in the earlier stages of mitral obstruction the left auricle dilates.

The left auricle frequently contains blood clots, partly of ante-mortem, partly of post-mortem, origin.

The condition of the left ventricle is controlled by circumstances of a character exactly opposite to those which obtain in regard to the left auricle. In consequence of the reduction in the amount of blood which passes from the auricle to the ventricle, the latter has necessarily a smaller quantity to propel into the aorta, and it has as a result a smaller amount of work to overcome. It might naturally be expected, therefore, that the ventricle would undergo changes in a direction the converse of hypertrophy, and this is the result in many instances of uncomplicated mitral obstruction without cardiac failure. It is not surprising, however, to find that instead of retaining its normal size and thickness, or of being even reduced in both, the left ventricle undergoes hypertrophy. It has to be remembered that if there be any failure of compensation, the backward pressure thence resulting tells back upon the venous system, so that there is an obstruction to the passage of the blood from the arterioles into the venous channels; this in turn leads to an increased amount of the work thrown upon the left ventricle, and is probably the reason why in a considerable proportion of cases there is some hypertrophy of the ventricle. Another consideration must be admitted as a possible explanation of the hypertrophy of the left ventricle, and it is one which has been more particularly urged by Giuffré. The diastolic aspiration performed by the left ventricle must be hindered in mitral obstruction, and if diastole be regarded, as it is by many authors, as an active muscular process, it follows that a certain amount of hypertrophy will be produced by interference with it.

The right ventricle is almost invariably involved when mitral obstruction is present, because no matter how thorough may be the attempt at compensation by means of the left

auricle, there still remains a certain increased stress upon the pulmonary veins, capillaries, and artery, by means of which an increased amount of energy is demanded of the right ventricle. It therefore becomes hypertrophied, as was indeed thoroughly understood by Corvisart. If failure of the energy of the heart makes its appearance, dilatation ensues, and as a consequence, more or less permanent tricuspid regurgitation results, which leads in turn to implication of the right auricle, showing itself both by hypertrophy and dilatation as a general rule.

In most instances of mitral obstruction there are evidences of chronic venous stasis in the lungs. Dilatation of the pulmonary capillaries and brown induration are the common results observed. Rupture of some of the blood vessels is apt to occur with resulting hæmorrhage. If compensation fails venous stasis passes into œdema, and when the right side of the heart fails there is apt to be hydrothorax.

Brockbank has recently called attention to a very interesting fact:—that gall-stones are much more frequently found in mitral obstruction than in other conditions. His results are that while cases without heart disease have a percentage of 5·4 of gall-stones, cases of mitral obstruction show 21·8 per cent.

**SYMPTOMS.**—Mitral obstruction may be absolutely latent; there are many cases in which a lesion, quite insufficient to produce any retardation of the onward passage of the blood, may yet give rise to interference with the current such as may be able to produce some of the characteristic physical signs by which obstruction is recognised. In such instances as these, and they are by no means uncommon, there is obstruction which may be termed relative.

The earliest symptoms of mitral obstruction are connected, as might well be expected, with the pulmonary circulation. Inasmuch as the hindrance to the access of blood brings about disturbances in the pulmonary veins and their tributaries, interference with the aeration of the blood is an early symptom. Breathlessness is accordingly one of the first, as it is one of the most persistent, symptoms of mitral obstruction, when the equilibrium of the circulation is disturbed. It may only be present on exertion, the patient at other times suffering

from no inconvenience, but in those instances which present some lack of compensation the breathlessness is apt to be constant, and on exertion it becomes serious dyspnœa. Paroxysmal dyspnœa, not infrequently periodic, makes its appearance in a considerable number of cases. These are to be regarded as falling under the title of cardiac asthma.

As a result of the venous stasis in the lungs, there is a great liability to pulmonary affections such as broncho-pneumonia, and when the retardation of the blood current is more seriously interfered with, passive hyperæmia and consequent œdema make their appearance. To breathlessness there are therefore added such symptoms as cough, attended by the expectoration of sputum, very fluid in character and often containing blood. In consequence of the long-continued backward pressure, some of the blood vessels may rupture, and the pulmonary hæmorrhage so produced reveals itself by the expectoration of blood in a more or less unchanged condition, attended by the general symptoms of pulmonary hæmorrhage.

It is to be remembered that the bronchial veins, which end in the right auricle, are not involved unless the right ventricle has begun to suffer in consequence of the mitral lesion; as the bronchial arteries, nevertheless, do not supply as much blood to the walls of the bronchial tubes as in health, on account of the reduction of the general arterial pressure, the bronchial tubes are liable to pathological changes. When, in addition to reduction of pressure in the bronchial arteries, there is produced, by interference with the right ventricle, a condition of high pressure in the bronchial veins, chronic bronchial catarrh is inevitable.

Pain cannot be regarded as a frequent symptom of mitral obstruction. It nevertheless occurs in a proportion of cases, and it is more likely to be localised over the anterior chest wall with little tendency to radiate into the shoulder and arm. As a consequence of the reduction of pressure in the arteries, the brain often suffers. Insomnia and headache frequently present themselves in the course of the disease.

When the right side of the heart fails in consequence of mitral obstruction, there occur all the characteristic symptoms of pronounced tricuspid incompetence. There is œdema of all

dependent parts. The functions of the digestive viscera are perverted, so that there is gastro-enteric catarrh, feeling of weight in the region of the liver, enlargement of the abdomen if ascites should supervene, increase of breathlessness if hydrothorax should occur, alarming tendency towards syncope if hydropericardium be present, scanty urine containing albumin, and suppression of the catamenia.

The appearance of any patient presenting pronounced symptoms of mitral obstruction is often eminently characteristic. Even in the case of patients who have but little cyanosis there is a tendency towards a dusky flush high up upon the cheeks, between which and profoundly cyanotic phenomena there is every intermediate gradation. It would be erroneous, however, to allow such a statement to pass without remarking that this mitral facies is frequently absent, and there may be no obvious change in the external appearance of the patient. One of the most common forms of mitral obstruction is to be found in young women of purely chlorotic appearance. Pallor of the skin, blanching of the mucous membranes, dyspnœa, palpitation, constipation, and catamenial and nervous troubles are present. To this special variety particular attention has been directed by Petit.

The pulse in mitral obstruction is sometimes destitute of any special characters, but much more commonly it has some noteworthy features. During the early phases of the disease the only characters present are that the vessel is rather empty, and shows a degree of pressure somewhat below the normal, but it is perfectly regular and quite equal. A sphygmographic tracing taken at this stage may closely resemble many obtained in apparently perfect health.

Later on, however, in the development of the disease, the pulse manifests features showing much greater departure from the normal. The vessel becomes more empty and the pressure falls to a much lower level, while at the same time the rhythm becomes extremely irregular and the pulse waves are very unequal. A sphygmographic tracing taken at this period is often extremely characteristic, as is shown in Fig. 148.

On inspection of the neck some morbid appearances may be seen connected with the veins, but as these are the effect



of the tricuspid incompetence resulting from the mitral affection, consideration of them will be delayed until the tricuspid disease is considered.

The præcordia usually shows some diffuseness or indefiniteness of the apex beat, which is frequently displaced outwards to the left, and it may in addition give evidence of some pulsation to the right of the sternum as well as in the epigastrium.

Palpation sometimes furnishes no evidence of any particular importance, but in many cases it yields information of sufficient value to effect a diagnosis. No phenomenon may be perceived on the application of the hand, with the exception of diffuseness of the apex beat and increased pulsation to the right of the sternum, as well as around the xiphoid cartilage,

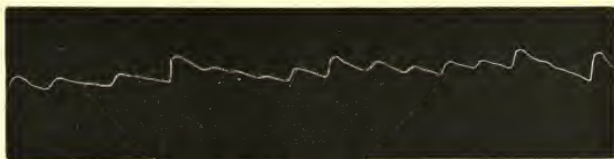


FIG. 148.—Tracing from the radial artery in a case of mitral obstruction ; pressure  $2\frac{1}{2}$  oz.

but in many cases a thrill is felt at the apex of the heart. This may be presystolic or diastolic in rhythm, and it is, with the exception of those few aortic cases previously referred to, pathognomonic of mitral obstruction. The sensation which is communicated to the hand is extremely well indicated by the name applied to it by those who studied it first in France, from its resemblance to the vibrations produced by the purring of a cat. The thrill is most commonly felt immediately before, and leading up to, the apical impulse. It is also quite common, however, immediately following the second sound ; and of very frequent occurrence, moreover, is a series of vibrations beginning with the impulse accompanying the second sound, and filling up the entire period until the occurrence of the apex beat. When this is the case the thrill may be found to wane somewhat during the post-diastolic period, and to wax again in intensity as it approaches the apex beat. It is obvious that there are three factors at work

in producing this thrill. There is, in the first place, the force, necessarily small in degree, exerted by the blood returning into the auricle by the pulmonary veins. There is, secondly, the aspiratory attraction produced by the active diastole of the left ventricle, and there is, thirdly, the energy produced by the contraction of the left auricle. The diastolic thrill is the result of the suction resulting from the active diastole of the left ventricle. The presystolic thrill is the direct consequence of the contraction of the left auricle; both of them are probably in part produced by the onward flow of the blood returning from the lungs, and in those cases presenting a continuous thrill, beginning with diastole and ending with systole, it is probable that this latter force fills in the interval between the diastolic and presystolic portion of the thrill. It is not at all improbable, however, that in such cases the systole of the left auricle begins somewhat earlier than is its wont, so that its commencement may immediately follow the cessation of the diastole of the ventricle. Rolleston points out that in some cases a vibration of the chest wall is caused by the action of an energetic heart on rigid ribs. He throws out the useful hint that, if there be any difficulty in determining whether an intra-cardiac origin is present or not, the fingers should be separated and placed in the intercostal spaces where the real valvular thrill is to be felt, while the osseous vibration disappears.

Cardiographic tracings taken from the apex beat in cases of mitral obstruction might be expected to reveal some important facts connected with this thrill. Much experience with this instrument has, however, failed in my hands to yield any profitable results. It might be thought that the various vibrations which go to make the thrill would be represented in a tracing taken from the apex beat. This, however, is seldom the case. The explanation is probably that the vibrations are in a great measure too subtle for our present means of investigation. An example of the tracing obtained by means of the cardiograph from a typical case is given in Fig. 24, p. 135.

Palpation frequently furnishes another important indication. At the base of the heart a very distinct impulse may

be detected attendant upon the closure of the semilunar cusps. This impulse is best felt over the second left intercostal space, and is produced by the increased pressure within the pulmonary circuit; it is of considerable importance from the point of view, not only of diagnosis, but also of treatment.

On percussion the area of cardiac dulness is sometimes absolutely unaltered. This is particularly the case when the obstruction is but slight, and the right heart therefore but

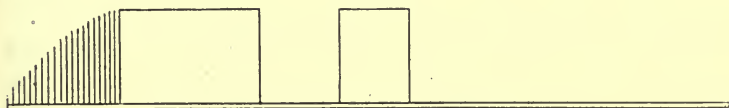


FIG. 149.—Presystolic murmur.

little involved. It is, however, more common to find that the transverse diameter of the area of dulness is more or less increased to the right as well as to the left—in fact the increase is usually greater towards the right than towards the left.

The facts ascertained by auscultation constitute the most characteristic feature in most instances of mitral obstruction. These consist in alterations in the rhythm and character of

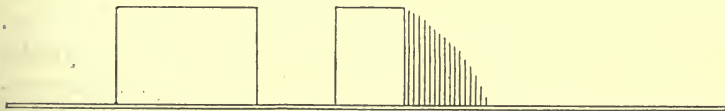


FIG. 150.—Diastolic murmur following second sound.

both cardiac sounds, as well as in the presence of abnormal sounds or murmurs. It may be stated as a general rule that those cases which present no characteristic murmurs of obstruction, but in which there are alterations in the character of the normal sounds of the heart, are more serious than those in which distinct obstruction murmurs are present.

In cases of pure mitral obstruction unattended by regurgitation, and uncomplicated by any disturbance of the right heart, or other result of failing compensation, the physical sign

found on auscultation is the characteristic murmur. This murmur may be presystolic, early diastolic, or late diastolic in rhythm, as shown in the accompanying figures. That is to say, it may be produced by the contraction of the left auricle, by the aspiratory force of the left ventricle, or possibly by the onward flow of the blood impelled by the energy of the right ventricle. This last factor cannot be regarded as so absolutely certain as the other two; yet it is extremely probable. The causation of the diastolic murmur of mitral obstruction has lately been the subject of an interesting paper by Rolleston.

The murmur is always rough in character. The same features which are translated by the sense of touch as a thrill give rise to the perception of sound, and, as in all other cases of this kind, when a thrill is perceived the sound which

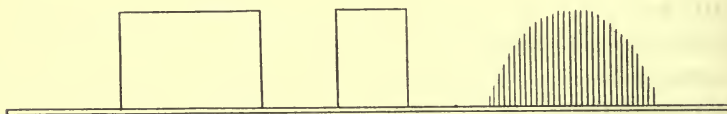


FIG. 151.—Late diastolic murmur.

accompanies it is harsh. The degree of harshness is not the same in the three different periods. The presystolic is almost invariably harsher than the diastolic murmur, and the early diastolic is always harsher than the late diastolic murmur.

Many controversies have arisen in regard to the presystolic murmur. These have, in so far as is necessary, been mentioned in the section dealing with murmurs in general, and require no further remark in this portion of the work.

The presystolic murmur usually terminates in a more or less accentuated first sound, and in instances of simple obstruction the sound is quite clear, being neither accompanied nor followed by any systolic murmur.

These murmurs are sometimes entirely absent, and many cases of mitral obstruction escape detection in consequence. It has already been seen that amongst cardiac murmurs those which are the result of mitral obstruction are much the most variable, and cases of pure obstruction of this orifice may at one time give a well-marked presystolic murmur, ending with



a loud first sound; at another there may be a characteristic diastolic murmur; and yet again a late diastolic murmur may make its appearance.

Mitral obstruction is very frequently accompanied by accentuation of the pulmonary second sound, or doubling of the second sound at the base, in which the pulmonic element is the louder. The explanation of this phenomenon has already been fully discussed in a former chapter of this work, and the subject need not again be entered upon. This doubling of the second sound undergoes considerable variation, not only in the intensity of the sounds and in the interval by which they are separated, but there is also a great tendency towards the disappearance and reappearance of the doubling.

In very many instances mitral obstruction fails to yield any characteristic phenomena on auscultation. As already seen, the presystolic and diastolic murmurs are extremely variable in their occurrence; their disappearance and reappearance are matters of everyday observation. The doubling of the second sound at the base of the heart, moreover, may also be absent, and even when it is present it is not of diagnostic importance, inasmuch as it may have its origin in some cause other than obstruction at the left auriculo-ventricular orifice. As a rule, in mitral obstruction the first sound is somewhat roughened, even when there is no evidence of a presystolic murmur, a subject which has already been described in a previous chapter.

DIAGNOSIS.—The recognition of mitral obstruction when its physical signs are fully present can never be a subject of any difficulty. The thrill, the murmur, and the doubling of the second sound, with pulmonic accentuation, present a triad of symptoms quite unmistakable. It is, however, far otherwise when all these fail; this they often do, for in the case of uncomplicated mitral obstruction there may be absolutely no evidence upon which a diagnosis may be based. As will be seen in dealing with combined mitral lesions, there is almost invariably sufficient evidence of both lesions, but in simple obstruction it is otherwise. Nevertheless, there is in most instances a degree of roughness about the first sound which throws some light upon the case, and this harshness in the

quality of the first sound, especially if associated with a double second sound, is often sufficient to justify the inference of obstruction.

It is difficult to say how it would be possible to determine in the case of double aortic murmurs associated with a presystolic murmur heard at the apex, whether this latter should be regarded as proof of a coincident mitral obstruction, or as being simply the aberrant murmur first noticed by Flint. The condition of the pulmonary second sound might possibly furnish some evidence. It must, however, be borne in mind that when there is obliteration of the aortic, there is no criterion by which to judge of the intensity of the pulmonary second sound. The state of the base of the lungs, and the condition of the right side of the heart, might also be helpful. Such points would require the most careful consideration, and might be really useful in diagnosis. This murmur has never come under my notice, or, as it might be better put, no presystolic murmur at the apex has ever been under my observation in which subsequent post-mortem examination has not revealed a mitral lesion; all this is accordingly pure speculation on my part.

Obstruction of the mitral orifice by means of a pedunculated thrombus attached to the wall of the left auricle has recently been described by Ewart and Rolleston. The rational symptoms and physical signs were identical with those of mitral obstruction and incompetence, and there could in such a case be no possibility of reaching a correct diagnosis.

PROGNOSIS.—In simple uncomplicated mitral obstruction the outlook cannot be regarded as very serious. In most instances where obstruction exists alone it is not very great, and it therefore interferes but little with the circulation. When obstruction is present in a higher degree it is almost invariably accompanied by regurgitation as well, and the prognosis in such cases must be considered elsewhere. Even in simple uncomplicated mitral obstruction the prognosis must always be given with reservation. There cannot be a doubt that any extra fatigue, physical or psychical, may in such cases produce a total breakdown of the circulatory equilibrium, which may be followed by fatal results. Furthermore, the patient who has any lesion of the mitral cusps is liable under the influence

even of apparently trivial affections to suffer from a lighting up of the valvular disease.

As contrasted with other valvular lesions, mitral obstruction may be said to be rather more serious than aortic obstruction; somewhat less serious than mitral incompetence; and very much less dangerous than aortic regurgitation.

It is hardly necessary to say that when compensation has been disturbed the outlook is much more serious, and symptoms showing that there is any implication of the lungs are to be regarded as warnings not to be overlooked. When œdema shows itself, or when the abdominal viscera are involved, the prognosis becomes even more serious.

TREATMENT.—In cases of mitral obstruction the great aim of treatment must be to obviate, in so far as may be possible, the tendency to pulmonary disturbances, seeing that the first symptom of failing compensation is an implication of the pulmonary veins. While therefore undertaking the treatment of mitral obstruction on the general principles applicable to all cases of valvular disease, the principal care in regard to this lesion must be devoted to preventing or removing any complications connected with the lungs.

The avoidance of sudden vicissitudes of temperature should be strictly enjoined, and if there be the least appearance of any pulmonary complication, it is well to confine the patient at once to a thoroughly ventilated apartment, with an equable temperature, and if necessary, to employ steam, either simple or medicated, as an inhalation. At the same time, even in those cases in which compensation is apparently perfect, it is advisable to employ *strophanthus* or *digitalis* in moderate doses, along with some ammoniacal stimulant. The further methods of treatment are those which have been described from the general point of view.

CASE 27. *Mitral Obstruction*.—B. R., aged 20, engaged in house-work, was admitted to Ward 25 of the Royal Infirmary, 1st February 1897, suffering from palpitation and breathlessness. There were no well-marked hereditary tendencies, and the patient's occupation and surroundings had always been satisfactory. At the age of 10 the patient suffered from chorea for about a month. About the age of 12 she frequently observed swelling of the ankles at night, but this entirely passed away and no



recurrence took place until recently. She had often been attacked by sore throat, but there had been no other rheumatic symptom. Five years before admission the patient's attention was attracted to breathlessness on exertion, often attended by palpitation. Both of these symptoms lessened on resting. In spite of medical assistance these symptoms remained, with occasional improvement and relapse, but during the few months preceding her admission she had been entirely incapacitated for any work.

On admission the patient was found to be 4 ft. 11 in. in height and 8 st. 11 lb. in weight. The temperature was normal. There were no obvious evidences of disease with the exception of a somewhat bright flush on the cheeks. The teeth were much decayed, the appetite was good, thirst was complained of. On examination of the abdominal viscera no abnormality could be detected. Slight enlargement of the lymphatic glands in the region of the jaws was obvious.

The patient complained of some pain in the region of the sternum if the surface was chilled. Palpitation occurred on the least exertion or emotion, and there was a sensation, sometimes as if the heart were turning over, sometimes as if it had stopped. Breathlessness always attended those symptoms, and was excited by the least exertion. There were no appearances of cyanosis connected with the mucous membranes. The radial artery had perfectly healthy walls. The pressure was low, the rate of the pulse 66 per minute, and the rhythm perfectly regular.

On inspection of the præcordia the apex beat was clearly visible outside of the left mammary line, five inches from mid-sternum. On palpation the impulse of the heart was felt to be of moderate intensity and preceded by a distinct purring thrill. The area of cardiac dulness extended two inches to the right and six to the left of the mid-sternal line, its upper border being at the lower level of the second left intercostal space. On auscultation in the aortic area no abnormal sounds were audible. In the mitral area there was a distinct murmur of mitral obstruction, commencing shortly after the second sound, waning somewhat in intensity and increasing again in loudness up to the first sound, which was distinctly slapping in character, but perfectly closed. This murmur was harsh in character. In the pulmonary area the second sound was much accentuated but not doubled. In the tricuspid area no abnormal sounds could be heard. The respiratory system revealed absolutely no symptoms of disturbance, and no other organ of the body was implicated.

By means of absolute rest and appropriate food, along with digitalis, she rapidly improved. This was followed in a week by strychnine and arsenic, and on 17th February the patient left hospital greatly improved in every respect.

This was a most excellent example of pure uncomplicated mitral obstruction in which compensation had been fairly well established but was liable to be disturbed. If the patient had been in a position in which it would have been necessary for her to go on with her work at all costs, there would inevitably



have been implication of the right side of the heart and a total breakdown.

CASE 28. *Mitral Obstruction with Anæmia and Leucocytosis*.—M. S., aged 20, a ward maid in the Royal Infirmary, had been from time to time under my observation, and was admitted to Ward 25, 6th January 1896, on account of breathlessness and debility. Her family history showed no facts bearing on the affection for which she was under treatment, and her social conditions had always been satisfactory. She had suffered from no disease which could in any way be connected with the affection in regard to which she sought advice. For some months she had suffered from the symptoms for which she was admitted.

On examination she was found to be 5 ft. 3 in. in height and 7 st. 13 lb. in weight. Her face was marked by a considerable degree of pallor; the lips, gums, and conjunctiva were also pale. The radial artery had healthy walls; it was fairly full and the blood pressure was moderate. The rate of the pulse was 78; its rhythm perfectly regular and the character of each pulse wave showed no departure from the health standard. There were no marked appearances connected with the veins of the neck or any other part of the peripheral circulation.

Inspection showed the apex beat to be in the fifth left intercostal space, three inches from mid-sternum. No other impulses were visible. On palpation, a rough thrill was found in the apical region, which began with the second sound and was continued up to the ventricular systole. The right border of the heart was found to be one and a half inches from mid-sternum, and the left was four inches from that point. On auscultation, a harsh murmur was heard in the mitral area, which commenced with the second sound and was continued, waning at first, and afterwards waxing in intensity, until it culminated with the first sound. The first sound itself was clear and unattended by any murmur. No evidence of any further abnormality could be detected in any other area, and there was almost no accentuation of the pulmonary second sound.

The condition of the patient calls for no further remark except in regard to the state of the blood, on investigating which it was found that the hæmoglobin amounted to 50 per cent. and the number of the red blood corpuscles to 3,000,000, while the white blood corpuscles reached 20,000.

Dr. Robert Muir kindly examined the blood along with me, and reported that the increase of the leucocytes more especially concerned the multinucleated forms, although eosinophile cells were also more numerous than usual. He regarded the condition as simply a leucocytosis, although the cause of the condition was not evident, as in cases of chlorosis the leucocytes are usually diminished in number. In this case the red blood corpuscles were somewhat altered in form, as often occurs in

cases of chlorosis. There were no nucleated red blood corpuscles and the blood plates were about the normal. It is quite possible that the case may be justly regarded as one of those linked with, if not dependent upon, chlorosis in some obscure pathological nexus. In this case the use of protochloride of iron and arsenious acid along with rest caused a rapid disappearance of the symptoms for which she was admitted to the ward, and in twenty-one days she was dismissed.

### MITRAL INCOMPETENCE.

Little need be said in regard to the history of our knowledge of mitral regurgitation. The lesion was anatomically recognised by the old authors to whom reference has so frequently been made, and the clinical recognition of the affection was due to the indefatigable exertions of the early auscultators of this century. The remarks of Laennec upon this subject are unfortunately most obscure, even in the later editions, as for example that which is so well known through the medium of Forbes' translation. With the work of Hope, the facts in regard to mitral regurgitation assumed definition and correctness, since he taught that regurgitation was denoted by a murmur attending upon the first sound. It must be admitted that his localisation of the murmur left a good deal to be desired, seeing that he stated that it was "louder opposite to the mitral valve (*viz.* at the left margin of the sternum, between the third and fourth ribs, *i.e.*, about three or four inches above the point where the apex of the heart beats) than elsewhere." From this time onwards gradual additions were made as regards the details of the clinical features of mitral incompetence, to some of which reference will be made in the sequel.

As in the case of the aortic, the mitral orifice is not provided with any means by which a reflux of blood can occur under strictly physiological circumstances. There is no provision whereby a safety-valve action can occur, such as is characteristic of the tricuspid valve. This fact was noticed last century by Hunter, and was emphasised long ago by Adams, who stated in his remarkable paper, "that the mitral valve so

perfectly closes the aperture of communication between the left auricle and ventricle, that in the natural state no reflux whatever is admitted. This (the reflux) so useful at the right side of the heart, would have been not only useless but injurious at the left side of the organ, as we find the general arterial system at all times equally ready to receive the blood during the systole of the left ventricle; and if the mitral valve did not perfectly close the left auriculo-ventricular aperture, a great deal of the force of the aortic ventricle would be wasted, whereby it would be incapable of moving the mass of blood which was destined to fill the arterial system. Pathologists, in looking to the different nature of the lining membrane at the two sides of the heart, as a means of explaining the greater liability of the left side to disease, have, perhaps, too much overlooked this circumstance, that while, from the unyielding nature of the mitral valve, all reflux into the auricle is prevented, from this very cause, which renders it effective in the circulation, is it exposed to more frequent injury from which organic disease may arise, and the ventricle to which it belongs becomes more liable to be ruptured by its own efforts." This most interesting passage, from a monograph which really laid the foundations for much of our present knowledge of cardiac pathology, is eminently worthy of consideration.

ETIOLOGY. — Mitral incompetence may be produced by changes in the cusps themselves or in the tendinous cords. Such alterations may result from endocarditis taking origin in the many different causes which have already been passed in review in dealing with that subject. It may, on the other hand, be the result of degenerative changes occurring in later life, or from long-continued strain. It may, further, be caused by traumatic agencies of direct or indirect origin, the result of which is to rupture some part of the valve, or tear some of the chordæ tendineæ.

The lesion is also produced by changes in the myocardium which interfere with the normal adaptation of the cusps. All causes which lessen the nutrition of the heart wall can in this way give rise to escape at the mitral orifice. Amongst such causes may be mentioned the febrile state, wasting diseases, anæmia, simple debility, myocarditis, and



muscular degeneration. It need hardly be added that muscular stress in such states is a determining factor. The incompetence may be brought about by interference with the muscular ring, or sphincter as it may be termed, whereby the normal systolic reduction in the size of the orifice is lessened, as was emphasised by Macalister. It may, moreover, be produced by dilatation of the ventricular cavity and disturbance of the functions of the papillary muscles, whereby the approximation of the segments is interfered with.

Mitral incompetence arising from those valvular lesions belonging to the first category is absolutely incurable. When produced by the causes belonging to the second group it is not infrequently a remediable disorder.

MORBID ANATOMY.—In those forms of mitral incompetence resulting from endocarditic processes there is almost, if not quite, invariably some contraction of the orifice, as well as incompetence of the valve—the combination, in short, of obstruction and incompetence. The cusps, therefore, present the appearances which have been already discussed in dealing with mitral obstruction. The lesions which produce the incompetence are shrinking of the cusps and fusion of their margins, frequently attended by the presence of vegetations.

Degenerative changes producing mitral incompetence do so by giving rise to rigidity and contraction of the cusps, resulting in deformity, and often associated with the different deposits, fibrous, fatty, and calcareous, which have been previously described. When produced by traumatic causes, mitral incompetence usually shows rupture of the chordæ tendineæ, or of the cusp close to the attachment of the tendinous bands.

The various causes which lead to mitral incompetence by acting on the cardiac muscle may do so in a threefold manner. There may be simply an enlargement of the auriculo-ventricular orifice by means of relaxation of the muscular structures surrounding it. Such a widening of the orifice must of necessity render it more difficult for the cusps to be adapted to each other, and thus an incompetence is produced.

An enlargement of the ventricular cavity may in the next place produce mitral incompetence in consequence of want of adaptation of the cusps, seeing that the ventricular walls may



become enlarged to a greater extent than the papillary muscles. In conditions of muscular relaxation it is quite obvious that this may occur, seeing that the relative extent of the ventricular wall as compared with the papillary muscle is so much greater. If the ventricular cavity enlarges to a greater extent than the papillary muscle, there must necessarily be a want of adaptation of the cusps.

In many cases these two different processes are found to be associated. A dilatation of the orifice is attended by an enlargement of the ventricular cavity, so that there is not merely a wider opening than can be closed by the cusps, but there is also a diminished power of adaptation from the disturbance of the relations between the papillary muscles and chordæ tendineæ, on the one hand, and the size of the cavity, on the other.

It must ever be remembered that a good many of the causes and effects which have just been considered are associated together. It is very common to find shrunken cusps fused together, and not merely studded with vegetations but altered by degenerative processes and associated with an enlargement of the orifice or even of the ventricular cavity.

The effects produced upon the heart by mitral incompetence are seen mostly in the left auricle and on the right side. The condition of the left auricle depends largely on the amount of regurgitation. When the regurgitation is free, the left auricle is found to be very considerably dilated, as well as hypertrophied. If, however, the regurgitation is not excessive, the condition is more like that which has been seen in mitral obstruction; *i.e.*, there is more hypertrophy of the auricular wall than dilatation of the cavity. The condition of the right side of the heart is entirely dependent on the amount of interference with the passage of the blood through the lungs, and it therefore bears some relation to the degree of regurgitation, and the consequent changes in the left auricle. The right ventricle is usually at once dilated and hypertrophied. The relative degree of dilatation and hypertrophy appears to be conditioned mostly by the nutritive possibilities of the heart.

The condition of the left ventricle is a matter which has been much disputed. It might at first sight appear that the

left ventricle should not undergo any considerable alteration in consequence of a change in the mitral orifice. Nevertheless, it has to be remembered that when regurgitation backwards into the left auricle is allowed, a larger amount of blood must be poured by the auricle into the ventricle during auricular systole, and the ventricle must therefore have a larger amount of blood within it at the commencement of systole. Its work is therefore augmented, so that, as is well put by Petit, "*le ventricule gauche subit indirectement le contre-coup de l'insuffisance.*" This appears to be the explanation of the common occurrence of dilatation and hypertrophy of the left ventricle.

The cavities on both sides of the heart frequently contain ante-mortem and post-mortem clots deposited upon the walls and amongst the tendinous cords, to which they are sometimes found firmly adherent.

Dilatation of the radicles of the pulmonary veins is almost invariably present, and in consequence of the long-continued stress to which they are subjected, the ramifications of the pulmonary artery and pulmonary veins are often found to be atheromatous. As results of these changes in the pulmonary vessels, more distant effects are found in the lungs. Chronic venous stasis leads to a condition of hypertrophy and œdema, in which the lung presents a close superficial resemblance to the spleen, and the presence of hæmorrhages is extremely common. The liver, the spleen, and the kidneys undergo the various changes consecutive to cardiac failure, mostly in the direction of fibrous increase, giving hardness and toughness, and the hollow viscera are found in a state of chronic venous enlargement with catarrh of the mucous membranes. The heart itself, when not undergoing fatty changes, is also the seat of fibrous increase. The brain has venous hyperæmia with œdema and effusion.

**SYMPTOMS.**—Mitral incompetence is often latent. So long as the heart is possessed of reserve energy, and is therefore able to restore the disturbed balance of the circulation, the existence of compensation prevents the development of any functional disturbances. It is otherwise when the equilibrium is lost, when various subjective and objective symptoms obtrude themselves.

Uneasiness, sometimes amounting to pain but more commonly giving the sensation of weight, is experienced in the region of the præcordia. Such sensations are not commonly complained of while patients are at rest, but they make themselves manifest on the slightest exertion.

Breathlessness is a very common symptom, and it also is chiefly found on exertion. Cough is extremely frequent in mitral incompetence, and in those cases characterised by cardiac failure the cough is attended by a watery sputum.

Nausea is often experienced, and occasional vomiting is its result.

Sensations of faintness and of giddiness, often accompanied by feelings of fulness in the head, or even by pronounced headache, are of common occurrence in this disease, and it is above all others that which is most likely to give rise to insomnia, and even brain symptoms such as illusions, hallucinations, and delusions.

The victim of mitral incompetence frequently shows the same appearances as have already been mentioned in regard to mitral obstruction. The dusky flush upon the cheeks, the cyanotic hue of the lips, ears, and nostrils, the arborescent capillaries upon the surface, the congested aspect of the eyes,—all are present in this disease and are somewhat more marked than in obstruction. A certain degree of jaundice is quite common in mitral incompetence, and when associated with cyanosis it produces a striking green shade of the complexion. Œdema of the dependent parts of the body coming on at night in the less severe cases, but persistent in those which are more serious, is seldom absent.

In uncomplicated mitral regurgitation the pulse may have little trace of departure from the normal standard, and sphygmographic curves may present a comparatively natural appearance, as shown in Fig. 152. The chief alterations undergone by the pulse lie in the direction of reduced pressure without much interference with the rhythm. In the later stages of the affection, however, and in those cases of organic incompetence when it is associated with obstruction, the pulse becomes empty, its pressure is low, its rate is accelerated, and its rhythm disturbed. In such instances the typical mitral



pulse may make its appearance, characterised by simple intermittence or extreme irregularity along with inequality of the pulsation.

Examination of the neck frequently shows venous pulsation due to interference with the right side of the heart.

The præcordia may present no morbid phenomena on inspection. It is, however, on the other hand, a very common occurrence to find the apex beat displaced outwards to the left. The impulse may be diffuse, and its situation, therefore, rather difficult to determine. There is not infrequently a distinct movement from above downwards in the third, fourth, and fifth intercostal spaces, and heaving in the epigastrium is of common occurrence. In this place reference may be made in



FIG. 152.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral incompetence; pressure 2 oz.

passing to the pulsation in the second left intercostal space, which is regarded by Naunyn and Balfour as the result of regurgitation from the left ventricle into the left auricle; the consideration of this subject, however, will be taken up when the different myocardial changes are discussed.

On placing the hand over the præcordia the cardiac systole may be found to present very different forms of impulse. Most commonly it produces a somewhat diffuse and rather feeble beat, often followed by an extremely sharp impulse accompanying the second sound, produced by the increased pressure within the pulmonary artery. Uncomplicated mitral incompetence is never accompanied by any thrill. This is a point to which particular care has been devoted by myself, and during many years' experience of this disease no case has ever presented itself for my observation in which a thrill was perceptible. Petit describes a systolic vibration commencing



with the apex beat and accompanying the entire systole almost up to the second sound, and he attributes it to the vibrations, produced under the energetic contraction of the ventricle, by the blood wave passing through the orifice into

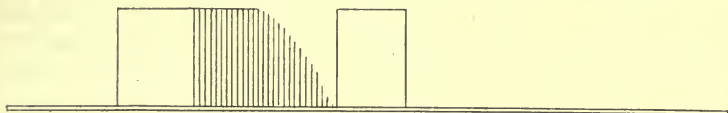


FIG. 153.—Systolic murmur following first sound.

the auricle. Such a phenomenon has never presented itself before me.

Percussion of the cardiac area reveals an enlargement laterally in both directions. The left border of the heart is enlarged outwards, so that it may extend beyond the mam-

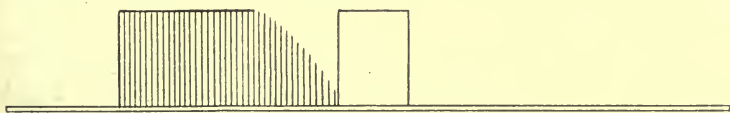


FIG. 154.—Systolic murmur accompanying first sound.

millary line, while the right border passes considerably farther outwards than in health. It is probable that the enlargement in both directions is due to the dilatation and hypertrophy of the right side of the heart, which causes it therefore to be projected farther over towards the left.

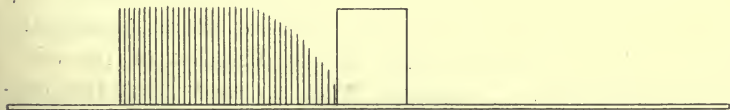


FIG. 155.—Systolic murmur replacing first sound.

On auscultation in the mitral area the characteristic murmur, following, accompanying, or replacing the first sound, is heard with its maximum intensity at that point. The rhythm of each variety is shown in Figs. 153, 154, and 155. The character of this murmur varies immensely, being sometimes low, soft, and blowing, and at other times high, rough,

and rasping. From the differences in the sound there is sometimes a tendency to draw certain conclusions as to the probable nature of the incompetence, yet he would be a somewhat bold observer who ventured to predict with too much refinement the exact nature of the lesion probably to be found. Nevertheless, in cases of mitral incompetence produced by organic changes in the cusps, the murmur is generally higher in pitch and harsher in quality—therefore presenting characteristics which may be summed up in the word shrill—than is the case in regurgitation resulting from muscular relaxation. The point of maximum intensity of the murmur of mitral incompetence is almost invariably at or close to the apex beat. It is propagated from this region in every direction, but to a different extent; for as has been shown in a previous section of this work, it is conducted to a greater distance in the direction of the axilla and scapula. In many instances it can be heard throughout the whole chest, as well over the right as the left side.

A murmur often heard outside of the pulmonary area, in the left second intercostal space, is regarded by Naunyn and Balfour as one of mitral incompetence propagated upwards from the seat of origin, at the valve, by means of the auricle to the appendix. This will be found discussed in a future chapter.

The position of maximum intensity of the systolic murmur in mitral incompetence is not altogether easy to explain. The close proximity, however, of the ventricular wall affords an easy means for the transmission of the sound, and it has to be remembered that the escape takes place through the cusps which are drawn down in the direction of the apex. The observations of Bergeon tend to show that the conduction of the murmur towards the apex is in part due to the eddy of the blood between the valvular cusps and the ventricular wall. Very frequently the murmur is found to have a position of intensity almost as great as that over the apex beat at a point between the left shoulder-blade and the vertebral column, and there cannot be the shadow of a doubt that the conduction of the murmur to this point is due to the proximity of the left auricle.

The intensity of the murmur presents many variations. It occasionally is only an accompaniment of a well-marked first sound. At other times it entirely replaces the sound, and apparently these different appearances are produced by the relation existing between the strength of the muscular wall and the condition of the incompetent cusps.

The second sound in the pulmonary area almost invariably undergoes changes in mitral incompetence. It may be merely accentuated, or it may be doubled, and as will be described in the subsequent section dealing with the affections of the pulmonary valve, there may be a diastolic murmur of escape through the pulmonary orifice from high pressure.

DIAGNOSIS.—The diagnosis of mitral incompetence is always an easy matter, but, while the mere determination of regurgitation can never present any difficulty, it is not so simple to deal with the differentiation between incompetence due to a valvular lesion and that due to muscular relaxation. It may be said, as a general rule, that when there is any evidence of obstruction as well as incompetence, the latter is due to an organic change in the valves, and the presence, therefore, of any of the features already detailed as pointing to the presence of mitral obstruction is of use in this regard.

It is sometimes stated that doubling of the second sound is pathognomonic of obstruction, and that its presence always indicates in cases of mitral incompetence the existence also of some valvular occlusion. This, however, as has already been stated previously, is an erroneous conception, and doubling of the second sound is not to be regarded as being of any diagnostic import.

PROGNOSIS.—The prognosis in mitral incompetence depends upon so many different circumstances that it is by no means easy to deal with. Simple mitral incompetence of slight degree may exist for many years without appreciable interference with health. The difficulty comes in, however, on attempting to determine in any given case whether the amount of regurgitation is small or large. The evidence afforded by physical examination is not altogether unmistakable, and the persistence of good general health and freedom from pulmonary symptoms, in spite of the existence of mitral incom-

petence, will be found to afford a better basis upon which to rest a hopeful prognosis than any results of investigation.

The absence of any symptoms of venous stasis, the freedom of the right side of the heart from any serious implication, the existence of an energetic cardiac impulse, and the presence of a loud first sound, are elements in any individual case which should justify a hopeful prognosis; and it may be added that, as a general rule, the outlook is more favourable in those cases in which a loud systolic murmur is present than in those in which an extremely feeble murmur can be detected. The existence of fair arterial pressure, and regular as well as equal pulsation, are also to be regarded as favourable appearances. Excessive frequency of the pulse is, as in almost all other conditions, a feature of the gravest import.

Mitral incompetence commencing in early life is necessarily of greater severity than when it is established at a later period, since it interferes with the processes of development and leads to disturbance of the general nutrition.

TREATMENT.—It often happens that the physician is placed in a position which allows him to observe the development of mitral incompetence. This occurs when the lesion takes its origin in some febrile affection. In such circumstances, by the exercise of care a considerable amount of influence may be exerted upon the future of the disease. By means of absolute rest, fluctuations in blood pressure may be avoided, as far as possible, while the use of general tonic remedies will, by acting through the blood, increase the nutrition of the cardiac muscle; in this way much may be done to avert the mitral change and thus to obviate the consequences to which it leads.

The conditions are analogous, in many instances leading to mitral incompetence by means of malnutrition, and the exercise of caution in such states will prevent the development of mitral incompetence. Such cases depend entirely upon the muscular apparatus; they will, accordingly, be described in dealing with the myocardium, and are only mentioned here in order to show their relationship to valvular lesions.

When mitral incompetence has been fully developed, the general principles of treatment should be based upon the con-



sideration of its principal consequences as it affects the lungs and the right side of the heart. Every care must be exercised in order to prevent, so far as is possible, interference with the circulation through the lungs, and every means adopted which will increase the aeration of the blood. Full details in regard to the treatment of mitral incompetence have already been given in the section devoted to the general treatment of valvular affections.

CASE 29. *Mitral Regurgitation*.—J. M., aged 13, schoolboy, was admitted to the Royal Infirmary, 12th December 1892, complaining of pain in the region of the heart. His father and mother, as well as a family of four brothers and sisters, exclusive of the patient, were in excellent health. The patient's environment had always been in every way satisfactory. His health was perfectly good until October 1891, at which date he suffered from rheumatic fever, and was confined to bed for three months. The pains at that time affected all his joints, but were unattended by any cardiac complications.

About a week before his admission he began again to suffer from pain. It made its appearance somewhat suddenly, and was rather severe. On cross-examination he admitted that from time to time since his recovery from the acute rheumatic attack, he had felt it, but never severely. It was, in his opinion, always intensified by carrying any heavy weights, such as he was used to do in his mother's shop.

On examination of the patient there were absolutely no obvious morbid phenomena of any description. The alimentary system appeared to be healthy, and there was no enlargement of any of the glands. No dropsy occurred even when the patient was up all day. The pain from which he suffered was felt over the præcordia, rather to the left of the mesial plane, and it extended upwards to the left shoulder. The neck afforded no abnormal movements. The patient was distinctly pigeon-breasted. The movements of the heart were visible in the third, fourth, and fifth left intercostal spaces. The radial artery was soft and elastic; the vessel was moderately full, and its pressure was about the average for the patient's age. The pulse rate was 88; it was perfectly regular and equal, and there was no undue celerity or tardiness as regards the individual pulse-waves. Palpation showed that the apex beat was in the fifth intercostal space  $2\frac{3}{4}$  in. from mid-sternum; the cardiac dulness extended  $1\frac{1}{2}$  in. to the right and 3 in. to the left of that line at the level of the fourth cartilages. On auscultation, the second sound in the aortic region was found to be rather louder than in the pulmonary, and hence might be regarded as slightly accentuated. There was no doubling of the second sound. In the mitral area there was a soft-blowing systolic murmur propagated towards the axilla, but audible for a short distance around the apical region. The first sound in the tricuspid region was absolutely free from any accompaniment and was well closed.

There were no morbid phenomena connected with any other region of the body.

The little patient, after treatment for a few days by means of strophanthus and complete rest, was put upon moderate doses of iron, and in the course of a short time was so relieved of all his symptoms that he could be dismissed.

This case affords an excellent example of the difficulty in determining whether regurgitation at the mitral orifice is due to some valvular lesion, or to relaxation of the sphincter of the orifice. There could be no question of dilatation of the heart, seeing that it was of such moderate dimensions, and therefore the diagnosis could only be that of relative incompetence through relaxation of the structures surrounding the orifice, or from the valves themselves.

It seems to me that in such a case as this an absolute decision is almost impossible. Had there been much accentuation of the pulmonary second sound, or a doubling of that sound, the diagnosis would probably have been that of combined obstruction and incompetence. It is, indeed, possible that in this case such a double lesion was present, but its absolute determination was beyond the possibility of certainty. It is probable that such a case as this, in which it was possible there was some endocarditic lesion of the cusps, would be reported as mitral incompetence, and it is an excellent illustration of the liability to error in statistics.

*CASE 30. Mitral Incompetence with slight Escape at the Tricuspid Orifice.*—J. D., aged 25, housemaid, was admitted to Ward 27 of the Royal Infirmary, 13th October 1897, complaining of breathlessness and weakness. Her father was well; her mother died at the age of 45, of bronchitis; one sister died of acute rheumatism; the other members of the family were well. Her environment had been healthy, and her duties not unduly severe. In regard to her previous health it may be said in a word that it had been satisfactory.

For some months she had been gradually losing strength and energy, and at length acted on a recommendation to enter the Royal Infirmary.

On admission she was found to be pallid in complexion with little colour in the mucous membranes. The appetite was indifferent, but the digestion was not very faulty, save for some constipation. The tongue was pale, but free from fur; the teeth were good; the abdominal viscera of their normal size. The hæmoglobin amounted to 45 per cent., and the red blood corpuscles numbered 3,500,000. The spleen, thyroid gland, and lymphatic glands showed no change.

No morbid phenomena could be observed in the neck or præcordia. The radial artery was soft and yielding ; it was rather empty, and its pressure low ; the pulse rate was 80 ; the rhythm was regular, and the pulsations equal. The apex beat could be felt somewhat diffusely in the fourth and fifth left spaces ; its chief impact being  $3\frac{1}{2}$  in. from mid-sternum in the fourth space. The upper border of the cardiac dulness was at the superior edge of the third left cartilage ; at the level of the fourth ribs it extended  $1\frac{1}{2}$  and 4 in. to right and left of mid-sternum. On auscultation there was weakness of all the cardiac sounds, and two soft-blowing murmurs were present. One had its maximum intensity in the mitral area, from which it was carried a short distance in all directions ; the other, a very gentle, nay, almost imperceptible, murmur, could only be heard at the inner end of the third left rib. There was no accentuation of the pulmonary second sound, and no doubling was present. A continuous venous hum was heard in the neck.

The facts regarding the other systems call for no remark, except that there was some scantiness of the catamenia, which, however, were regular. By means of rest and protochloride of iron along with arsenic the patient speedily recovered.

It was clear that in this case the chief condition was one of anæmia, and the point of greatest interest lay in the fact that the cardiac dilatation mainly affected the left ventricle. As will be shown in another chapter, dilatation of the heart from anæmia usually implicates both ventricles at once, but the right is, as a general rule, more affected than the left. In this case the area of dulness showed that there was less enlargement of the right than the left side of the heart, and the distribution of the murmurs corroborated this fact.

## MITRAL OBSTRUCTION AND REGURGITATION.

The combination of obstruction and incompetence is always, as would naturally be expected, due to organic disease, and is very much more frequent in its occurrence than either lesion singly.

ETIOLOGY AND MORBID ANATOMY.—Combined obstruction and incompetence may be produced either by endocarditic changes or degenerative processes, of which the former are considerably more frequently met with. The various factors which have been already considered in the production of endocarditis play their most important role in the evolution

of this double lesion. It is often possible to trace out directly the connection between cause and effect. It is also, however, a frequent experience to have no distinct evidence of the connecting link, in which case the lesion has been developed insidiously. Mitral obstruction and incompetence do not take their origin by any means so frequently from degenerative processes; but it must be allowed that in elderly people the combined mitral lesion occurs purely from such processes.

Examination of the heart in such cases shows in the main three distinct types of lesion. The cusps may have vegetations adherent to them, which produce at once some obstruction and at the same time bring about a maladaptation, whereby regurgitation is allowed. A further step in lesions of this type is found when the cusps have become shrunken and retracted, so that they can by no possibility close the aperture. The second great type is found in those cases in which the valves are thickened and often roughened, while they, as well as the tendinous cords, are harder and more resistant than in health. Here a certain amount of obstruction is present, and in consequence of rigidity the cusps cannot be adapted to each other, and incompetence therefore results. The third group includes all those cases in which the cusps become fused together so as to form a slit of small dimensions, which from the infiltration or deposition going around it produces incompetence as well as obstruction.

The effects produced by the combined lesion are more serious than those which take place in either obstruction or regurgitation. The implication of the auricle is greater, and it therefore shows a considerable degree of dilatation as well as hypertrophy. In many instances dilatation alone is found, and the walls of the auricle are very thin. There is more tendency to backward pressure upon the pulmonary circulation, and more distinct implication of the right side of the heart, which, becoming interfered with at an earlier period of the disease, allows greater disturbance of the systemic veins. The effects, therefore, upon all the internal viscera are more pronounced.

**SYMPTOMS AND DIAGNOSIS.**—The clinical features produced by the combined lesions show a blending of those properly



appertaining to each; but the combination gives rise to more marked symptoms than does either disease by itself. It is needless to traverse the ground already gone over as regards the general symptoms and physical signs; but it may be remarked that, with the greater disturbance not merely of the pulmonary, but of the systemic circulation, the clinical features of the combined lesion are much more marked than in simple obstruction or incompetence. As regards evidences of pulmonary hyperæmia dilatation of the right ventricle, oedema of the dependent parts, enlargement of the solid viscera within the abdomen, and catarrhal conditions of the mucous membrane, the combination is swifter in effecting their production than is either lesion singly.

The pulse is much more likely to become irregular at an early period in the development of the disease. Venous pulsation or turgescence in the neck, and epigastric pulsation in the abdomen, are frequently found. The size of the heart as ascertained by percussion is considerably exaggerated, and on auscultation the combined murmurs may be heard. It is here that the chief element of difficulty is apt to step in; seeing that the presystolic and diastolic murmurs arising at the left auriculo-ventricular orifice are the most fugitive of all, it is no wonder that some uncertainty occasionally arises. On auscultating a case of combined obstruction and incompetence, it is very common indeed to find that no murmur is to be heard with the exception of the systolic mitral murmur. Along with this, however, there is almost invariably a roughness in the character of the first sound, and the well-known remark that this often sounds as if it were the "b" of the syllable "rub," of which the previous part had disappeared, is singularly apt. Accentuation of the pulmonary second sound is usually more marked in the case of the double lesion when only one aspect of it, whether obstructive or regurgitative, is found. The existence of considerable irregularity in the cardiac pulsations, along with a rough first sound passing into a systolic murmur, and followed by an accentuation of the pulmonary second sound, may be regarded as significant of the double lesion. It is unnecessary in this place again to refer to the fact that occasionally a presystolic murmur may

have its origin without mitral obstruction. This subject has been already thoroughly discussed.

**PROGNOSIS AND TREATMENT.**—There cannot be the least doubt that when obstruction and incompetence are united, the prospect for the patient is much more gloomy than when there is only obstruction or incompetence. From the greater severity of the effects, there is much more disturbance of every function connected with the nutrition of the heart, and the prognosis is therefore much graver than in either single lesion. Although, as a rule, free from the intensely painful sensations experienced in many cases of aortic disease, it nevertheless happens that from combined obstruction and incompetence at the mitral orifice there is more long-continued distress than in any other valvular combination. Little need be said in regard to treatment, as it is practically that which has been enjoined for the individual lesions by themselves. The management, however, requires to be carried out more sedulously than in the case of either of the lesions by itself.

**CASE 31. *Mitral Obstruction and Regurgitation.***—W. N., aged 8, school-boy, was admitted to Ward 22 of the Royal Infirmary, in March 1897, suffering from breathlessness and palpitation. His father was in good health. His mother died at the age of 31 of mitral disease. The patient had one brother and one sister—both well.

He had never been robust, but had not suffered from any serious affection until the present illness began. Its commencement was quite insidious, the little patient gradually beginning to feel ill and lose strength some months before his admission. Dyspnoea had for some time been constant, and there had been some swelling of the legs and other dependent parts of the body.

On admission the patient was found to be pale and bloodless in appearance, with a small bright flush on either cheek. The alimentary system yielded no symptoms of any importance. The spleen was not enlarged. The pulse varied from 96 to 112 during the first few days of his residence in hospital. The vessel was compressible and moderately full. The pulsation was perfectly regular and equal, and each pulse wave, although not large, was well sustained. There were no abnormal phenomena connected with the neck or præcordia on inspection. The apex beat was in the fifth intercostal space 3 in. from mid-sternum. On applying the hand a well-marked thrill was ascertained at the apex beat. The thrill obviously commenced with the diastolic recoil, and ran on, first diminuendo and then crescendo, to the systolic impulse of the heart. On percussion, the right and left borders of the heart at the level of the fourth costal cartilages were respectively  $1\frac{1}{2}$  and  $3\frac{1}{2}$  in.

from mid-sternum. On auscultation in the aortic region, a faint blowing systolic murmur could be heard, but on passing downwards it was found to be propagated from below. In the mitral area there was a systolic murmur accompanying, but not obliterating, the first sound. It was soft and blowing in character, and was propagated in every direction to some extent, but more especially towards the axilla. The second sound was immediately followed by a rough diastolic murmur which gradually diminished in intensity, and this became louder as it passed into a very harsh presystolic murmur running up to the first sound. In the pulmonary area the second sound was obviously accentuated, but not to any very great extent. Auscultation of the tricuspid area revealed the existence of a soft-blowing systolic murmur. There were a good many crepitations at the bases of both lungs posteriorly. The other systems call for no remark, as they presented no abnormal phenomena.

There could be no difficulty in diagnosing obstruction of the mitral orifice, and incompetence of the mitral and tricuspid valves. The chief interest in the case which has just been narrated lies in the fact of the hereditary predisposition to disease exhibited by the patient.

*CASE 32. Mitral Obstruction and Incompetence.*—T. R., aged 39, compositor, frequently consulted me as an out-patient at the Royal Infirmary on account of breathlessness and cough. He stated that he was unaware of the presence of rheumatism at any time among any of his relations. His mother, nevertheless, died about two years before he presented himself at the Infirmary, of heart disease. This may, however, have been some senile form of cardiac affection. His father died of apoplexy about twenty years before the patient came under observation. He himself had suffered from scarlet fever on one occasion, and had several times been attacked by sore throat. Eighteen months before coming under observation he began to be breathless, and a few months later a persistent cough developed. These symptoms gradually became worse until he applied for relief at the Infirmary.

On examination, a slight tinge of jaundice was observable along with some lividity of the lips. His teeth were carious and the tongue was furred, yet the appetite was good and the other digestive functions excellent. The abdominal viscera were normal in size.

The patient complained very much of constant palpitation, of frequent faintness, and of occasional giddiness. Dyspnoea was constant. The radial walls were healthy; the vessel was moderately full, and of fair pressure. The pulse was extremely irregular in rhythm, but its rate was always under 60, being usually about 52. The character of the pulsation was tardy. On examination of the neck, well-marked pulsation could be seen in the jugular veins. There was a degree of prominence of the præcordia, more especially in the region of the fourth left costal cartilage. The apex beat was in the fifth intercostal space. On palpation there was a



long rough diastolic thrill at the apex beat, of which no evidence could be obtained with the cardiograph. On percussion the upper limit of cardiac dulness was at the third left costal cartilage. The right border extended  $1\frac{3}{4}$  in. to the left of mid-sternum, while the left extended  $3\frac{1}{2}$  in. outwards at the level of the fourth rib. On auscultation in the aortic area a double second sound could be heard. In the mitral area a soft systolic murmur accompanied the first sound, and there was a rough purring murmur beginning with the second sound and lasting over more



FIG. 156.—Tracing from the apex beat in Case 32.

than half of the long pause. In the pulmonary area a soft systolic murmur was perceptible, but this was propagated from below. The second sound was distinctly doubled, and the later part of it was the louder in this area. In the tricuspid area there was a soft-blowing systolic murmur, which on being followed up was found to be the same as that heard in the pulmonary area. There were no symptoms or physical signs connected with the lungs or urinary organs. He had for some time been suffering considerably from insomnia. The patient improved under treatment, and was able to attend to his duties for two or three years. Finally, however, a systole set in, of which he died.

The chief interest in this case lay in three points. The hereditary tendency requires notice in the first place, linked with the insidious origin of the disease. The absence of any presystolic, and the extreme roughness of the diastolic, murmur are also worthy of attention. The case, further, seemed to be one in which a cardiographic tracing might be expected to yield some evidence of the vibrations. A tracing was accordingly taken from the apex beat by means of a direct cardiograph, but, as can be seen in the accompanying figure, it throws but little light upon the condition.

**CASE 33.** *Mitral Obstruction and Regurgitation of Insidious Origin.*—C. W., aged 13, schoolgirl, was admitted to the Royal Infirmary, 5th



May 1894, complaining of breathlessness. The patient's father had suffered for some time from chronic renal disease, her mother from some chronic cardiac lesion. She had four brothers and six sisters, who were in excellent health. Her social conditions had always been good. The patient's previous health had been satisfactory, and she had in particular suffered from no rheumatic symptoms, or any infectious disease likely to produce endocarditis. Two years previous to admission she began to suffer from shortness of breath, more particularly on exertion, and soon afterwards she had pain in the left side of the chest, which was more frequent after any excitement or exercise. These symptoms were succeeded by a winter cough, which troubled her during the two following winters, and giddiness had also been a frequent symptom.

The patient was found to be somewhat small for her age, and her appearance suggested a slight degree of bloodlessness. The tongue was clean and moist, and no digestive symptom called for any remark, except occasional sickness. The lips were somewhat pale, as well as the gums and conjunctivæ.

Pain, dyspnoea, and palpitation always followed exertion. The pain was situated over the præcordia, but did not extend towards the left shoulder. There were no morbid phenomena to be observed on inspection of the thorax; but a distinct pulsation of auricular rhythm was present in the veins of the neck on both sides. The radial arteries were healthy, the vessels fairly full, showing moderate pressure. The rate of the pulse was 90. It was perfectly regular, and the pulsations absolutely equal. Palpation revealed a distinct thrill which could be felt to accompany the diastolic rebound of the heart. The apex beat was in the fifth intercostal space,  $2\frac{1}{2}$  in. from mid-sternum. No other impulse was perceptible. The heart on percussion at the level of the fourth ribs had its right border  $1\frac{1}{2}$  in. and its left border 3 in. from mid-sternum. On auscultation, murmurs were heard accompanying the auricular and ventricular systole and the diastolic phase. There were obviously two systolic murmurs, one of which had its point of maximum intensity outside of the præcordia in the sixth left intercostal space; while the other was heard most distinctly just outside of the junction of the sixth left costal cartilage and the sternum. This systolic murmur blended at all intermediate points, yet its total area of audition only measured transversely  $4\frac{1}{2}$  in., and vertically  $3\frac{1}{2}$  in. The diastolic murmur, which was extremely harsh in character, had its point of greatest loudness at the apex beat, but it was heard over a wide area over the front and sides of the chest—an area measuring 12 in. transversely, and  $7\frac{1}{2}$  in. vertically. The presystolic murmur had its point of maximum intensity in the sixth intercostal space, but internal to, and somewhat lower down than, the maximum intensity of the mitral systolic murmur. The character of this murmur was harsh, but by no means so harsh as the diastolic murmur, and it was, moreover, short and sharp. Its extent of audibility was comparatively small, and it was conducted farther towards the mesial plane than outwards. The area over which it could be heard measured  $3\frac{1}{2}$  in. transversely, and the same vertically. The second sound was everywhere clear and sharp.

In this case the auscultatory cycle seemed to commence with the diastolic murmur, which immediately succeeded the second sound, without interfering in any way with it. This murmur, which was, as already said, extremely harsh, gradually rumbled to an end, and was succeeded by a brief pause before the short, sharp, presystolic murmur commenced. This latter terminated in a well-marked somewhat rough sound, to which succeeded the short blowing systolic murmur. The particular points in this case requiring comment are, the insidious onset of the disease and the special characteristics of the physical signs.

CASE 34. *Mitral Obstruction and Incompetence.* — N. T., aged 16, domestic servant, was admitted to Ward 25 of the Royal Infirmary, 19th February 1894, complaining of breathlessness.

Her father had died some years before the date of her admission, but the patient was unable to throw any light upon the cause of his death. Her mother and one brother enjoyed excellent health, and there had been no other members of the family. The patient's social conditions had always been excellent. As regards her previous health, it did not appear that she had suffered from any serious affection, and in particular there was no history of rheumatism. Nine months before her admission she began to suffer from want of breath, which increased, and was accompanied also by some swelling of the ankles at night, so that she was led to present herself as a patient at the Royal Infirmary.

The alimentary and blood glandular systems called for no remark. There was no cyanosis, and almost no appearance of anasarca. The radial artery was soft and elastic, of moderate fulness and pressure. The rate of the pulse was 70, its character rather bounding, and its rhythm regular. No abnormality was observed in connection with the neck or præcordia, until, on placing the hand over the latter, a distinct thrill was perceived. It was purely presystolic in rhythm, and was situated at the apex beat, which occupied the fourth intercostal space 3 in. from mid-sternum. The right border of the heart was  $2\frac{1}{4}$  in. and the left 4 in. from mid-sternum. On auscultation, two distinct murmurs could be made out. A harsh presystolic murmur with its maximum intensity at the apex beat, and its conduction spread over an oval area measuring  $7\frac{1}{2}$  in. horizontally, and 6 in. vertically. The systolic murmur had its point of maximum intensity also at the apex beat, but its extent of audibility was considerably less, measuring  $5\frac{1}{2}$  in. horizontally, and 4 in. vertically. The pulmonary second sound was somewhat accentuated, and this was the only other symptom of disturbance which could be found. Beyond a few scattered crepitations throughout the lungs, there was no other morbid appearance anywhere, and a few days of rest and strophanthus allowed the patient to leave hospital with considerable improvement of all her symptoms.

The most interesting point in connection with this case is the fact that the presystolic murmur manifested such a much larger area of audibility than the systolic.

CASE 35. *Mitral Obstruction and Incompetence.*—J. C., aged 44, plasterer, was admitted to Ward 22 of the Royal Infirmary on 16th April 1894, suffering from breathlessness and palpitation. His father died at the age of 72 of apoplexy, and his mother at the age of 40 of dropsy. Two sisters, the only other members of his family, were quite strong. He had been married, but his wife had died after the birth of twins—a boy and girl, the former of whom was still-born, while the latter had grown up to be a healthy girl. His social conditions had been satisfactory. His previous record in regard to health showed that he had undergone two attacks of rheumatic fever, and that he had laboured several times under pulmonary congestion. The illness for which he came under treatment began, about the New Year previous to admission, with palpitation and breathlessness, and as treatment produced no beneficial effects, he sought admission to the Infirmary. There were no symptoms connected with the alimentary or hæmopoietic systems. There was some swelling about the ankles, and some duskiness in the colour of the lips, nostrils, and ears. The radial arteries were rather hard, and both as regards pressure and fulness they exceeded in some degree the normal limits. The rate of the pulse was 80, and it was perfectly regular. No abnormal appearance could be observed in connection with the præcordia or neck. The apex beat was imperceptible to touch. The heart on percussion was found to extend  $1\frac{1}{2}$  in. and 4 in. to the right and left sides of the mid-sternum at the level of the fourth rib. On auscultation, the heart sounds were extremely weak, more especially the first sound in the mitral and tricuspid regions; while the pulmonary second sound was slightly accentuated. The bases of the lungs showed abundant crepitations; there was no abnormality connected with the urinary system. After a few days of perfect rest, and the administration of strophanthus and ammonia, the patient's condition obviously improved, and on auscultation a systolic murmur made its appearance close to the probable situation of the apex. It was soft and blowing in its character, and day by day increased in intensity. At first confined to the region of the apex, it gradually extended in all directions until it could be heard over the entire præcordia and a short distance below it, while at the same time it began to manifest two different points of maximum intensity. One of these was in the fourth intercostal space  $3\frac{1}{2}$  in. from mid-sternum, and the other close to the junction of the fifth and sixth costal cartilages with the left edge of the sternum. At this latter point, the murmur was much higher in pitch than in the former. While this change was going on two other murmurs began to be perceptible at the probable apex. These were presystolic and diastolic in rhythm. The former of these was situated below and a little to the outside of the maximum intensity of the systolic murmur, and was 4 in. from mid-sternum. Its area of audibility was small, occupying an oval-shaped area measuring 4 in. by  $2\frac{1}{2}$  in a diagonal direction,

passing upwards and outwards from the sternum. The latter was situated a little to the outside of the point of maximum intensity just described, and at a slightly higher level. It was distinctly systolic in rhythm and was propagated upwards, inwards, and outwards, so as to give rise to an irregularly oval-shaped area, measuring  $4\frac{1}{2}$  in. by 4 in. Both these murmurs were harsh and rough in quality; but the diastolic was decidedly harsher than the presystolic.

The chief point of interest in this case, apart from the somewhat singular distribution of the murmurs, which form a well-marked contrast to those of the preceding case, lies in the absolute lack of any murmurs on the admission of the patient, and their gradual evolution after the heart had gained strength through appropriate treatment.



## CHAPTER XII.

### AFFECTIONS OF THE PULMONARY ORIFICE.

SOMETIMES termed, according to Balfour, "the region of romance," the pulmonary area has been, nay, still is, the subject of much discussion. Some of the debates which have been connected with this area find no fitting place in a description of lesions of the pulmonary orifice, and will be more suitably discussed in another section. The present chapter must be devoted solely to the diseases of the pulmonary orifice and its cusps.

Lesions of the pulmonary valve have been recognised anatomically since the description of an instance of this affection by Morgagni, but the clinical features presented by diseases of the orifice and cusps were not elucidated until long afterwards. Hope laid down rules for the recognition of pulmonary stenosis and incompetence in the first edition of his work, but does not seem by that time to have met with a case of the latter lesion.

### PULMONARY OBSTRUCTION.

As already seen, this is one of the most frequent congenital heart affections. It is quite otherwise with regard to the lesion as developed after birth, for pulmonary obstruction is one of the most uncommon cardiac diseases arising in extra-uterine existence. In the remarks which follow, the congenital form of the affection will, so far as possible, be left out of account, and attention will be directed, except in so far

as is specially mentioned, to the class of acquired pulmonary obstruction.

Our knowledge of obstruction of the pulmonary orifice taking its origin during the independent existence of the patient, is in great part due to the original observations and industrious researches of Philhouze, Paul, Solmon, Duguet, and Vimont, the last mentioned of whom has collected a large number of recorded and unrecorded cases.

**ETIOLOGY.**—Acquired pulmonary obstruction is usually the result of endocarditis, the existence of which at the pulmonary orifice has been established by the work of several authors, amongst whom may be more particularly mentioned Meynet and Mayer. Endocarditis confined to the pulmonary orifice was reported by von Wahl, and a somewhat similar instance, in which, however, there was a spread of the lesion to the cardiac walls, is also on record. The cause of the endocarditis is often obscure, but in many instances it has been found to be one of the acute affections. Acute rheumatism is by no means so commonly linked with the origin of this disease as with the production of endocarditis of the other valves, yet it must be credited with a certain number of cases. The eruptive fevers are likewise responsible for the production of pulmonary endocarditis in several instances. Specific infection has been described in rare cases as producing obstruction by the development of gummata. Direct injury may cause obstruction, as in a case narrated by Dittrich, in which, from the kick of a horse on the præcordia, obstruction was rapidly developed. When all these different cases are taken into consideration there nevertheless remains a considerable number of cases in which the etiology is absolutely obscure.

**MORBID ANATOMY.**—The obstruction may be entirely produced by a change in the pulmonary cusps. This, according to Vimont, is by far the most frequent form of obstruction, occurring in 22 out of 32 cases. The most frequent lesion is the union of the cusps at their edges, so that a more or less funnel-shaped arrangement is produced, with its apex continued upwards into the pulmonary artery. When this is the case, the valves are always thickened and usually some-

what indurated; they may even be absolutely rigid from calcareous deposits. In such cases there is almost invariably incompetence as well as obstruction. Instead of, or in addition to, lesions having this character, vegetations may be found upon the cusps. When vegetations are associated with other lesions of the cusps, the obstruction may be extreme. The vegetations, however, may be the sole lesion, and they may be present without any incompetence. They give the ordinary features of valvular changes as seen elsewhere.

Another form of obstruction is produced at a somewhat lower level, in part of the conus arteriosus. This is the "*rétrécissement préartériel*" of Paul. It appears to be due in every instance to ventricular endocarditis. In obstruction situated at this level, the walls of the conus arteriosus are dense from the presence of fibrous tissue, and, as might readily be expected, the constriction is somewhat unequal in most instances. This form of obstruction may, apparently by extension, be associated with lesions of the cusps, and it is not uncommon to find a communication between the right and left ventricles when obstruction is situated in the conus arteriosus. According to Paul it takes its origin in myocarditis, arising by extension, and resulting in loss of substance of the septal wall.

Pulmonary obstruction may, further, take its origin in endarteritis of the pulmonary artery. Within recent years, attention has been particularly directed to obstruction taking its origin in this way. It must, nevertheless, be stated that in cases of long-continued interference with the pulmonary circulation in consequence of disease of the lungs, as is noticeably the case with regard to emphysema, the obstruction of the artery from endarteritis is by no means a common occurrence.

**SYMPTOMATOLOGY.**—Pulmonary obstruction is usually accompanied by some dyspnoea, but this breathlessness does not seem in most instances to reach any considerable degree. It seems, in fact, as in analogous instances of aortic obstruction, that the ventricle is able, by means of hypertrophy, to overcome the obstacle in great part.

It has been the general belief that obstruction of the

pulmonary orifice produces a considerable amount of cyanosis. This certainly is the case in congenital obstruction, but the opinion expressed by Paul certainly agrees with my own limited experience of this lesion, that, in acquired pulmonary obstruction, there is but little tendency to cyanosis; probably this is to be explained in the same way as absence of dyspnoea until cardiac failure makes its appearance.

There is no great tendency to venous stasis, and there is, therefore, but little œdema of the legs or interference with the abdominal viscera. My own observations do not accord with the experience of Paul in regard to the appearances presented by the fingers and toes, as in the few cases seen by myself there has been a quite evident tendency to clubbing of the fingers and toes, with enlargement and arching of the nails.

From the cases which have been hitherto collected, there can be but little doubt that acquired pulmonary obstruction, like the congenital form, produces a liability to phthisis pulmonalis.

On examining a case of pulmonary obstruction, the radial pulse is found to present no special peculiarity unless the muscular walls of the heart are failing. There may be venous turgescence or venous pulsation in the neck, but such phenomena are by no means inevitable accompaniments.

Inspection of the præcordia, as a rule, presents nothing abnormal, unless there be a slight outward extension of the apex beat, and some pulsation in the epigastrium. On applying the hand over the base of the heart, a distinct thrill, systolic in its rhythm, may be felt, and sometimes this thrill extends over a considerable portion of the præcordia. The area of cardiac dulness may be slightly enlarged transversely. The most important evidence obtained by physical examination, however, is that furnished by auscultation. The systolic murmur at the base of the heart is the special sign of the lesion. This murmur has, in almost every recorded instance, had its maximum intensity at the sternal end of the second left intercostal space. It is accurately systolic in rhythm and may continue almost to the second sound. Its character is, as a rule, harsh, but this feature is subject to variation in individual cases. There is one prominent characteristic about



this murmur—the superficial note which it seems to possess. This systolic murmur is propagated over a considerable area of the chest, but it is never heard in the great arteries of the neck. Its rhythm is shown in Fig. 157.

DIAGNOSIS.—The determination of pulmonary obstruction depends almost entirely upon the fact that the systolic murmur is not propagated into the cervical arteries. The mere position of the maximum intensity of the murmur goes for comparatively little, and it is the direction in which the murmur is conducted that is of the greatest importance. Changes in the cavities of the right side of the heart might be expected to occur in this affection, and, as a matter of fact, hypertrophy of the right ventricle has been from time to time recorded. This, however, is an observation founded more upon the

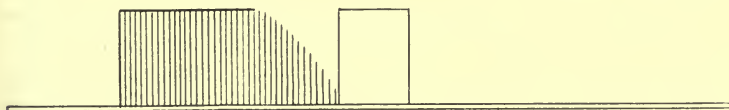


FIG. 157.—Systolic murmur accompanying first sound.

results of post-mortem investigation than clinical observation, and but little value can be assigned in diagnosis to any enlargement of the right side of the heart.

The existence of any clubbing of the fingers is a strong point in favour of the diagnosis of pulmonary obstruction.

The greatest difficulty in diagnosis lies in the differentiation of obstruction at the aortic and pulmonary orifices. As already said, the position of the maximum intensity of the systolic murmur is of comparatively little moment; it is its conduction which is of vital importance. In many a case of aortic obstruction the murmur is heard most distinctly to the left of the sternum, but in all such cases it is conducted into the carotid and subclavian arteries. It is otherwise in the case of pulmonary obstruction; the murmurs here are not propagated into these arteries.

Patent ductus arteriosus presents some features closely resembling those of pulmonary obstruction, with which it is, as a matter of fact, often associated as a congenital lesion. The

essentials by which patent ductus arteriosus may be distinguished are, that the systolic murmur has its point of maximum intensity at least two inches from mid-sternum, and, that it is continued beyond the second sound. This last fact is of the greatest importance, because such a murmur can by no possibility be produced at any of the arterial openings; it can only have its origin in an artery in which the blood pressure does not fall immediately on the cessation of ventricular systole.

PROGNOSIS.—In cases of pulmonary obstruction the outlook is not favourable, since most of the recorded cases have been cut off at an early period of life. The development of pulmonary tubercle is in many instances the cause of death. In other cases, however, death has taken place in consequence of cardiac failure.

TREATMENT.—The management of pulmonary obstruction requires attention to the general rules to be followed in the treatment of valvular lesions, with special reference, however, to the case of the lungs. It is, therefore, more particularly necessary to watch over the ventilation and warmth of the chamber in which the patient is treated. If there be any symptom of bronchial or pulmonary trouble, the employment of warm moist air, which may be medicated by means of resinous or balsamic substances, will be found advantageous in addition to the use of cardiac stimulants.

CASE 36.—A. S., aged 22, factory girl, was sent to me on 10th February 1896, in the out-patient department of the Royal Infirmary, by my colleague Dr. Maddox, with the information that she had been attending the ophthalmic department for embolism of the retinal vessel, and with the request that a report should be furnished in regard to the state of the heart. The patient's father and mother were both in excellent health. She had only had one brother, who died as a baby; two sisters were perfectly well, but two had died during infancy and one in child-bed. Until the illness for which she had sought admission, the patient had enjoyed excellent health.

During the month of November she became bloodless and breathless, and was unable to continue her occupation. About a month previous to the date when Dr. Maddox sent her to see me, she suddenly became conscious that the sight of the right eye had undergone a diminution in its clearness, and she thereupon consulted him on the subject.

On examination the patient's condition was obviously so unsatisfactory

that she was strongly advised to come into the Infirmary, in order to undergo thorough treatment, and in consequence she was admitted to Ward 25, on 17th February 1896.

On her admission the patient was found to be extremely pale, but her nutrition was in no way defective; she was, in fact, quite plump. Her height was 5 ft.  $\frac{3}{4}$  in., and her weight 7 st. 2 lb. The alimentary system furnished no symptoms of any abnormal condition. Examination of the blood showed that the hæmoglobin was 20 per cent., and the number of red blood corpuscles was 2,300,000 per c.mm.

There was slight œdema of the ankles, but the most characteristic peculiarity connected with the extremities was a considerable degree of clubbing of the toes as well as of the fingers, with distinct arching of the nails. The radial pulse was 94 per minute; the vessel was healthy and the pressure was low; the rhythm regular and equal; there was nothing approximating to the Corrigan type of pulsation. There were no abnormal phenomena connected with the neck. The apex beat could be seen in the fifth intercostal space, four and a quarter inches from mid-sternum. On palpation, no abnormal pulsation could be detected, but there was a distinct thrill at the base to the left of the sternum. On percussion, the right border of the heart, at the level of the fourth rib, was two inches, and the left, at the same level, four and a half inches from mid-sternum. At the fifth rib the left border was five inches from mid-sternum. On auscultation, loud murmurs could be heard everywhere with the systole and diastole; but these murmurs, on careful examination, were found to have their greatest loudness at the base of the heart. The systolic murmur had its point of maximum intensity at the upper edge of the manubrium sterni rather to the left of the middle line. The diastolic murmur was loudest opposite the third costal cartilage and slightly to the left of the middle line. From these points the two murmurs faded away gradually in every direction. A very soft murmur was present during the cardiac systole in the carotid arteries.

On examination of the respiratory system, there was an impaired percussion sound over the bases of both lungs, and a diminution of the respiratory murmur and vocal resonance, but there were no adventitious sounds. The urinary system yielded no symptom of importance. There had been amenorrhœa since Christmas. The nervous system gave no evidence of any disturbance with the exception of the ocular phenomena in the right eye.

The consideration of this case, which has been utilised for another purpose in a previous chapter, left, to my mind, no doubt as to its being one in which there was a double lesion at both the aortic and pulmonary orifices. The former was rendered probable by the fact that although there was no water-hammer character in the pulse, or capillary pulsation, there was yet no clear evidence that the aortic cusps closed, as there was no distinct second sound anywhere. As above

mentioned, the systolic murmur was propagated up the arteries of the neck, which was proof of an aortic obstruction, seeing that no dilatation of the aorta could be thought of. The latter was also, to my mind, absolutely clear, in consequence of the clubbing of the fingers and toes, as well as on account of the fact that the systolic murmur was quite different from, and far harsher in character at its point of maximum intensity than, the sound which was propagated into the arteries of the neck. The reason why this case is placed under the heading of pulmonary obstruction is, that it appeared to be much more an instance of obstructive lesion than of incompetent valves. The further details will be found on p. 430.

### PULMONARY INCOMPETENCE.

The earliest instance on record of pulmonary regurgitation is that mentioned by Chevers, which was not, however, diagnosed during life. In the fourth edition of his work, Hope describes a most interesting case, in which, with other lesions, pulmonary incompetence was a prominent feature. Although a large number of cases have been placed on record since the date of these observations, the detection of pulmonary incompetence is still surrounded by serious difficulties. In the following pages it is not my intention to enter upon the historical aspect of the subject. This has been rendered unnecessary, seeing that, in one of the most valuable contributions to the study of cardiac disease during recent years, Barié has not only followed the development of our knowledge of this particular lesion, but has also given a brief statement of the facts recorded in 58 cases, two of which were observed by himself. His memoir on this subject will, for future workers, remain a storehouse of clinical and pathological facts, for which he deserves the cordial thanks of all interested in diseases of the circulation.

ETIOLOGY.—Pulmonary incompetence is certainly an affection of early life; according to the researches of Barié, its greatest frequency is between the ages of 18 and 34. It occurs frequently as a congenital affection, but, in this case,



it is always accompanied by obstruction of the orifice. It occurs with equal frequency in both sexes.

The most frequent exciting causes are rheumatism and acute infectious diseases, more especially the eruptive fevers.

Degenerative processes giving rise to sclerosis and atheroma have been occasionally described, and this form of etiology has been attributed, with more or less reason, to chronic alcoholism.

**MORBID ANATOMY.**—Congenital lesions of the pulmonary cusps, such as anomalies in their number or alterations in their form, have already been referred to in the section dealing with congenital heart disease, and require no further remark in this place.

Pulmonary incompetence as a disease acquired during the separate existence of the individual, shows for the most part the results of endocarditis or of degeneration. The cusps are usually thickened and indurated, as well as shrunken in size and distorted in form. There are occasionally ulcerations upon the surface of the cusps, or they may be perforated by larger or smaller apertures.

The orifice of the pulmonary artery is in some instances found to be dilated. It is more common, however, to find that it is contracted, as in the accompanying illustration, Fig. 158. In other instances the orifice is possessed of its normal calibre.

The secondary results upon the heart are those of hypertrophy with dilatation of the right ventricle, and usually of the right auricle also. There is frequently some fatty degeneration in the muscular substance of the right ventricle. Secondary lesions are found in various internal organs affected by interference with the return of blood to the heart, and its proper aeration in the lungs, but these present no features of particular interest.

In some instances there exist, along with pulmonary incompetence, other lesions, such as communications between the two auricles, or between the two ventricles, or patency of the ductus arteriosus. When such structural alterations are present they will inevitably lead to the conclusion that the pulmonary lesion is probably of congenital origin.

Relative or functional incompetence of the pulmonary cusps undoubtedly exists. There is a natural provision for regurgitation under certain circumstances at both the orifices on the right side of the heart, which appears to have been first observed by Hunter. .“The valves of the pulmonary artery,”



FIG. 158.—Pulmonary incompetence with obstruction, the cone diameter of the orifice being .85 in.

he says, “do not do their duty so completely as those of the aorta; and if we inject a pulmonary artery towards the right ventricle, it does not so completely hinder the injection passing into that cavity; nor are the two portions of injection completely separated, when the artery is injected from the ventricle, as in the left side. So far as respects injection, the same observations are applicable to the *valvula tricuspides*; therefore, I believe the valves of the right side of the heart are

not so perfect as those of the left ; from hence we may suppose that the universal circulation requires to be more perfect than that through the lungs." Little notice was taken of these observations until Adams investigated the subject. To him is due the merit of showing clearly that what is necessary towards the maintenance of the systemic would be injurious to the pulmonic circulation, where so many cases must temporarily retard the passage of blood through the lungs.

From the clinical point of view the first writer who dealt with this subject was Stokes, who in the following passage enunciates very clear views in regard to it. "There is another form of insufficiency of the valves which arises, not from disease of the valves themselves, but from dilatation of the cavities when carried beyond a certain point. It is probable that this condition will be found more frequently at the right side, where it may affect both orifices, and be attended with dilatation of the pulmonary artery."

Gouraud, in an interesting monograph on the influence of pulmonary affections on the right side of the heart, devotes a section of his work to relative incompetence of the pulmonary cusps, and in this he follows the views of Stokes.

In a contribution made by me to the study of affections of the right side of the heart, some years ago, while in ignorance of the observations of Stokes and Gouraud, the conclusions arrived at from some experiments on the pulmonary valves of the heart were given, and may be briefly quoted here. As the average result of a series of experiments made with a column of fluid, it was there stated "that from the semilunar valves of the pulmonary artery of the sheep a strong jet escaped until the column of fluid reached  $14\frac{1}{2}$  in., from which height it trickled until the valves became incompetent with a column of 9 in. In the ox a strong jet was emitted down to 12 in., and dropping of the fluid reduced the superincumbent column to the height of 6 in., when competence was established. In the healthy human heart a jet escaped down to 13 in., and the valves were competent with 8 in. of fluid resting on them. Now, in each case of the pulmonary valves, with a column of fluid exceeding 6 ft. in height, perfect competence was obtained in

a very simple manner by constricting the pulmonary artery. A cord tied round the artery, exactly at the attachment of the valves, gave the means of perfect control over the escape, so that, by varying the amount of tightening, the jet was converted into a drop falling quickly or slowly, and this in turn was totally stopped. The whole diminution of circumference only amounted to a few lines. This shows clearly that the escape is caused by distension of the elastic artery, and relative incompetence of the valves." It may be added that, tested in the same way, it was found that the aortic valves never allowed any escape, and the paper referred to goes on to say: "Such experiments show with certainty that the orifices of the right side of the heart are not closed so perfectly as those of the left, and point to the ease with which they probably allow escape with any supernormal pressure."

Although these observations convinced me that there was probably some escape at the valve whenever there was any considerable increase of pressure in the pulmonary artery, no case came under my notice, until comparatively recent years, in which there was absolute evidence in proof of the correctness of the view based on them; but in the interval other observers, who have successfully devoted much attention to the diseases of the circulation, have independently advanced the opinion that a diastolic murmur occurs in consequence of high pressure in the pulmonary artery.

In his little work on the heart, Graham Steell, after referring to the diastolic murmur of aortic dilatation, says: "I am inclined to believe that a murmur of similar mechanism occurs in the right side of the heart, when there is much obstruction to the pulmonary circulation with a dilated pulmonary artery."

Duckworth described a case of mitral and tricuspid stenosis in which there was a temporary pulmonary diastolic murmur, which he considered as due to the dilated state of the vessel. It disappeared before death, and at the autopsy the pulmonary cusps were not found to be markedly altered.

The whole subject was again reviewed more recently by Graham Steell, who proposes to call this diastolic murmur,



occurring independently of disease or deformity of the valves, "the murmur of high pressure in the pulmonary artery."

These views receive emphatic support from some remarks by Barr on mitral stenosis, in which, writing on the obstructive diastolic murmur of mitral stenosis, he says: "I am convinced that it is often confounded with a short, soft-blowing diastolic murmur, which not infrequently occurs in this disease at the pulmonic valve, and which arises from slight regurgitation into the right ventricle, owing to the high pulmonic tension."

The whole subject was fully discussed, four years ago, in a paper by myself, dealing with the diagnosis of pulmonary incompetence.

**SYMPTOMS.**—The affection might be expected to produce considerable influence on the functions of the lungs through interference with their blood supply, and no doubt this is to some extent in every case its effect; yet the degree of disturbance for which it is responsible is difficult to estimate on account of the extreme rarity of regurgitation except in association with some other lesion. It must produce some dyspnoea and cyanosis, and such special appearances have always been found in the cases which have been recorded. These symptoms, except towards the close of life, are in most cases only found on exertion. During repose they cause but little inconvenience to the patient.

When the closing scenes of cardiac failure make their appearance, the dyspnoea becomes continuous, and the patient presents a deeply cyanosed appearance. Such symptoms are frequently attended by cough and other evidences of lung disorders.

In consequence of stress, the right ventricle undergoes changes. It becomes dilated as well as hypertrophied, and regurgitation at the tricuspid orifice accordingly takes place. There is consequently early implication of the systemic venous circulation. Venous stasis in its various forms is inevitable, resulting in enlargement of the solid abdominal viscera, catarrh of the mucous membranes, anasarca of the dependent parts, and effusion into the serous sacs. In this affection, just as in pulmonary obstruction, clubbing of the fingers and arching of the nails are frequently to be seen.

The physical signs present in pulmonary incompetence are mainly those of dilatation and hypertrophy of the right ventricle, along with the evidences furnished by auscultation. There may be turgescence, or obvious pulsation of the veins of the neck. The radial arteries may yield no features of special import, yet the artery is, as a rule, but poorly filled and the blood pressure is low. The volume may be large or small, the pulse frequent or rare, and the rhythm regular or irregular. It is obvious that these conditions are not directly connected with the affection of the right side of the heart. The apex beat may be farther to the left than in health, so that its pulsation may be observed outside the mammillary line. A distinct pulsation may also be observed in the epigastrium, and this, moreover, has occasionally been seen in the inter-

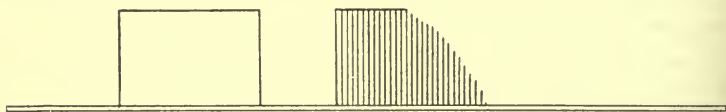


FIG. 159.—Diastolic murmur accompanying second sound.

costal spaces to the right of the sternum. Palpation gives further evidences of these facts, and reveals in some cases a thrill at the base of the heart, coincident with the second sound, and having its greatest intensity to the left of the sternum. The area of cardiac dulness is found to be increased both to the right and to the left. On auscultation a diastolic murmur is heard. This murmur may be diffused over a wide area, but it appears invariably to have its maximum intensity in the second left intercostal space. It is propagated towards the apex of the heart, and its line of conduction is chiefly to the left of the sternum. The character of this murmur varies considerably. It may be soft and blowing, or harsh and rasping. In those cases in which it is associated with a similar lesion of the aortic cusps, it is usually louder than the murmur produced by the latter. Its rhythm is shown in Fig. 159.

**DIAGNOSIS.**—The diagnosis of pulmonary incompetence should not present any great difficulties, yet it is a subject which requires a considerable amount of care. The affection

most likely to be confounded with it is aortic incompetence. At first sight it may appear extraordinary that the two lesions should ever produce symptoms which might be mistaken, seeing that the position of the murmur in each case should lead to correct conclusions, but, as has been shown by two illustrative cases of aortic disease (Case 25 and Case 26, pp. 514 and 517), the maximum intensity of the murmurs may be quite deceptive. The characteristic murmur of aortic incompetence is not infrequently to the left of the sternum, a fact which certainly should induce caution in effecting a diagnosis.

It might be thought that the conduction of the murmur would be of more importance than the mere position of maximum intensity of the murmur in each case. The propagation of the murmur, however, does not materially aid. There are, nevertheless, two points as regards murmurs, of some value. The diastolic pulmonary murmur is not propagated directly towards the apex, and if a murmur of this rhythm is heard distinctly at the apex of the heart, it is probably of aortic origin. A diastolic murmur, moreover, heard over the carotid arteries is proof positive of aortic incompetence, showing, as has previously been seen, such a degree of regurgitation as will allow of the generation of a murmur in the carotid. The presence of the characteristic Corrigan's pulse, and of capillary pulsation, must of necessity be regarded as most important evidence of aortic incompetence, while cyanosis, dyspnoea, and clubbing of the fingers must be allowed to weigh considerably in favour of pulmonary incompetence. All these, however, may occur in any valvular lesion from cardiac failure.

The chief difficulty in diagnosis as between aortic and pulmonary incompetence arises when both lesions are present in the same individual. In the patient whose case is recorded below (Case 37, p. 576) there could not possibly be any doubt that aortic disease was present, and the only uncertainty was as regards the presence or absence of a pulmonary affection. In such cases the maximum intensity and lines of conduction of the murmurs do not greatly help in the differential diagnosis. The other accompaniments afford evidence of higher value.

Patent ductus arteriosus is not likely to be mistaken for incompetence of the pulmonary valve, and the converse is also not likely to occur. The thrill and murmur present when the arterial duct remains open are of late systolic rhythm, and with care this fact is always easily determined; although the general symptoms are somewhat alike in the two conditions, more especially in regard to the presence of cyanosis and dyspnœa, the rhythm and the murmur and thrill should be sufficient to allow of an accurate diagnosis.

PROGNOSIS.—The prognosis in this valvular affection is, of necessity, always grave, inasmuch as, from the implication of the functions of the lungs, there is great liability to complications, and the further disturbance of the circulation tells fatally upon the right side of the heart. Bronchitis and broncho-pneumonia are especially likely in this way to aggravate the condition and to bring about a fatal result. It need hardly be added that when the disease exists in childhood whooping cough is a very dangerous addition. Frerichs pointed out that pulmonary tuberculosis is frequently found in association with this affection—a fact which has been discussed in the section on congenital disease.

TREATMENT.—The treatment in cases of pulmonary incompetence must be more particularly directed to the possibility of pulmonary complications, and prophylaxis will therefore have especial reference to those conditions which may induce or aggravate bronchial catarrh.

The evidences of unfavourable conditions, not merely as regards the external air but as regards the ventilation of the apartments occupied by the patient, will require close attention. Treatment must also have especial reference to the condition of the lungs in every instance; apart from this, it is but an aspect of the general treatment of cardiac failure.

As an excellent example of combined obstruction of the orifice and incompetence of the valve, in which the latter was the predominant feature, associated with aortic disease, the following case deserves notice.

CASE 37. *Aortic and Pulmonary Disease*.—Charles W., baker, æt. 45, married, was admitted to Ward 22 of the Royal Infirmary, on 15th May 1893, complaining of breathlessness, and swelling of the feet and legs.



His father was 70 years old, and in good health. His mother had died at the age of 64, of bronchitis. Of three brothers, one was in good health, another had died in infancy, and the remaining one had been killed by an accident. Of five sisters, four were alive and well, but one had died in infancy. The patient's social conditions had always been fairly good. He had in early years enjoyed very good health; but latterly, in consequence of three attacks of acute rheumatism, he had never felt well. The illness for which he sought admission began, about four months previous to his entrance into the hospital, with breathlessness, which was followed by the development of swelling of the lower limbs.

The patient was pale, with bloodless lips and gums, but the pallor was to some extent masked by a certain degree of cyanosis. There was considerable œdema of the lower limbs. The fingers, as well as the toes, were clubbed at their extremities, and the nails were distinctly arched. The appetite was small and capricious. Constipation and diarrhœa frequently alternated. The teeth were much decayed; and the tongue, which was pale, was covered by a slight fur. The abdomen showed no ascites. The liver extended six inches in the mammary line. The hæmopoietic system presented no abnormal features save those of anæmia.

The radial arteries were soft and elastic, the vessels empty, the tension low; the rate of the pulse was from 70 to 80, it was perfectly regular, each pulse wave was of large size and short duration,—in short, the patient had a typical Corrigan's pulse. On producing a flush over the forehead by friction, it was seen to become paler and redder with the alternate systolic and diastolic phases.

On inspection of the neck, a violent pulsation was seen in the carotid arteries, as well as some undulation in the external jugular veins, while over great part of the præcordia there was an extremely distinct systolic impulse, which gave to the hand, however, the sensation of short duration. The apex beat was felt in the sixth intercostal space, four inches from mid-sternum. No thrill could be detected on palpation. The deep cardiac dulness in the left parasternal line began in the second intercostal space, nearer the second than the third cartilage; it extended, at the level of the fourth cartilage, from two and a quarter inches to the right, to four inches to the left, of the mid-sternal line. On auscultation, two murmurs were heard; one accompanied the first, the other replaced the second sound. Both of these murmurs were loudest at the base: the systolic, which was loud and rough, having its point of maximum intensity at the junction of the third left costal cartilage with the sternum; while the diastolic, which was quite as loud, though somewhat less rough, had its point of maximum intensity over the third left costal cartilage and the adjacent part of the fourth intercostal space, one inch and a half from mid-sternum. Both murmurs were heard with diminishing intensity over the entire præcordia,—the systolic extending farther downwards and to the left; the diastolic slightly farther upwards and to the right. A slightly increased intensity of the systolic murmur could be discovered both in the mitral and tricuspid areas, and a distinct systolic

murmur was heard in the carotid arteries. The distribution of these murmurs is shown in Fig. 160.

The patient was very breathless, and coughed a good deal, the cough being accompanied by a frothy serous expectoration. The only physical sign connected with the respiratory system was a rather coarse crepitant râle over the bases of both lungs. The urine, which was small in amount, contained a trace of albumin. The patient was restless and sleepless, but those symptoms were in great part due to the dyspnœa.

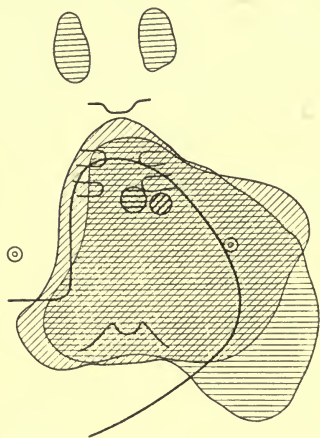


FIG. 160.—Pulmonary systolic and diastolic murmurs.

The diagnosis in this case presented, in one respect, some difficulty. There could be no doubt that there was stenosis of the aortic orifice—the propagation of a systolic murmur up the carotid arteries was sufficient proof of this; and there could also be just as little question in regard to the fact of aortic incompetence, since there was the characteristic water-hammer pulse, and the significant capillary pulsation.

The considerable hypertrophy of

the left ventricle also supported this. The point round which some difficulty hung was with regard to the condition of the pulmonary orifice and its valves. The position of the maximum intensity of the murmurs seemed to indicate a probability that both obstruction and incompetence existed, and this seemed the more likely, seeing that there was some cyanosis, with distinct clubbing of the fingers and toes. On the other hand, the small degree of cyanosis might have been the result simply of cardiac failure, and if this had recurred from time to time, it might also have given rise to the change in the ends of the phalanges. It was my opinion that, while the diagnosis of aortic stenosis and incompetence was absolutely established, and while admitting the probability of a pulmonary lesion, it was better to leave it an open question whether such an affection was present. It seemed to me, however, extremely likely that, if the pulmonary valves were healthy, there was such a pulmonary regurgitation, as will be described in the sequel, produced by a functional inability of the pulmonic cusps to close completely, in consequence of the high pressure within the pulmonary circuit.

In spite of every method of treatment, the patient became steadily worse. The dependent parts developed more œdema; ascites and hydrothorax came on; the urine fell off in quantity, and contained more albumin; and the dyspnœa and cyanosis increased. Death occurred quietly on 7th July.

The post-mortem examination was performed the following day by Dr. Leith, whose notes are appended.

*External Appearances.*—Rigidity absent. Lividity marked. Considerable dropsy.

*Thorax.*—There were extensive old adhesions in both pleural sacs.

*Heart.*—The pericardium was adherent all over; the adhesions being slender, and easily broken through. They were extensive both in front and behind, chiefly consisting of fine threads attaching the two surfaces together. There were two large irregular nodulated masses of osseous consistence: one to the right of the pulmonary artery, which was the larger, about the size of a hazel nut; the other to the left of the artery. Both were very irregular in shape, very jagged and nodular on their external surfaces, and lay in both layers of the pericardium, the two layers being here inseparable. The pulmonary valves were incompetent, as were also the aortic. The cone-diameter of the pulmonary was .85 in., and of the aortic .6 in. All the pulmonary cusps were thickened. This was especially marked at their attachments, which were almost cartilaginous in consistence. The lunules also showed considerable thickness, although not so pronounced. The condition was one of well-marked chronic endocarditis. There were some recent vegetations at the junction of the anterior and right posterior cusps. At the right margin of the right posterior aortic cusp there was an irregular perforation larger than a crow-quill; the margins were irregular and stained pinkish. Above it, and slightly to the right, there was a small aperture in the commencement of the intima of the aorta, *i.e.* just at the upper part of the sinus of Valsalva. This aperture led into an aneurysmal sac a little to the right, about the size of a bean. The left ventricle was considerably dilated, its wall being but slightly thickened. The muscle was somewhat paler than usual, and besides some fatty change was probably the seat of a chronic myocarditis. There was some fatty change in the wall of the right ventricle, and its muscular substance was somewhat hypertrophied. The cone diameter of the tricuspid orifice was 1.3 in. The cusps were slightly but uniformly thickened. The cavity of the right auricle was dilated, and its walls a little hypertrophied. The cone diameter of the mitral orifice was .88 in. The cusps were chronically thickened, the anterior markedly so. The thickening was not uniform. It was greatest at the contiguous margins of the valves. The wall of the left auricle was hypertrophied, but the cavity was not dilated. The heart, with the pericardium, weighed 1 lb. 10 oz.

*Lungs.*—Left weighed 1 lb. 15 oz. There were old pleuritic patches over both lobes and between them. There was marked œdema in both lobes, and brown induration in the lower lobe. Right weighed 1 lb. 14 oz.; it showed old pleurisy at the base, the apex, and between the lobes. There was œdema and congestion of all the lobes.

*Liver* weighed 3 lb. 6 oz. The gall-bladder contained blackish green bile, somewhat inspissated. The liver substance was firm. The surface was granular towards the anterior margin. On section, there was a nutmeg



condition present, with considerable fatty change of the peripheral portion of the lobules, but not much bile-staining.

*Spleen* weighed 14 oz., and showed some perisplenitis. It was large and firm. On section, the Malpighian bodies were distinct, and stood out as white points upon a dark purple. There was no reaction with iodine.

*Kidneys*.—The left weighed  $7\frac{1}{2}$  oz., and was firm. On section, it was pale. The superficial cortex showed irregular areas, and was diminished in places. There was a cyst about the size of a pea in it. The capsule stripped off badly. There was some superficial cirrhosis, and some slightly waxy change. The right weighed  $6\frac{1}{2}$  oz. It resembled its fellow.

*Stomach*.—The stomach contained some brownish fluid, and showed a chronic catarrh, but was otherwise healthy.

The result of the post-mortem examination in this case showed it to be possible to be too cautious in the diagnosis of rare lesions.

Functional pulmonary incompetence, shown by the diastolic pulmonary murmur of high pressure, is most excellently illustrated by the three cases with which this chapter may fitly conclude.

**CASE 38.—*Functional Pulmonary Incompetence.***—Maggie G., æt. 18, engaged in household duties, was admitted to Ward 25 of the Royal Infirmary, on 5th June 1893, complaining of pains in her wrists and elbows. Her father and mother, both aged 42, had always been in good health. She had four brothers and one sister, all very strong, but three brothers had died in infancy. The patient's social surroundings had always been good. She had never been very robust, and four years before admission had suffered from a rheumatic attack, since when she had never felt quite well. About four months before entering the hospital, pains had begun in the joints, and had persisted ever since.

On her admission, the patient was found to be somewhat pale, with a bright spot on each cheek. The skin was moist. The tongue was slightly furred, but the digestive system was otherwise healthy. There was no symptom connected with the hæmopoietic system.

She complained of some palpitation and a slight degree of breathlessness. The pulse was of low tension and moderate volume, perfectly regular, and varying in rate from 80 to 90. There was some pulsation in the veins of the neck, and a very distinct impulse in the second left intercostal space. On palpation, the apex beat was found to be in the fifth left intercostal space,  $3\frac{1}{4}$  in. from mid-sternum. The pulsation, systolic in time, in the second left intercostal space, was found to be most distinct  $1\frac{1}{4}$  in. from the mid-sternal line. A tracing obtained from it by means of a revolving cylinder is given in the accompanying figure (Fig. 161). No thrill could be detected over any part of the præcordia. The



cardiac dulness extended 1 in. to the right, and 4 in. to the left, of the middle line at the level of the fourth rib. On auscultation, a venous hum was heard in the neck, and there were murmurs, systolic in rhythm, over the whole præcordia, which on careful analysis proved to be twofold. Around the region of the apex beat, and with its maximum loudness in the fourth interspace  $3\frac{1}{2}$  in. from mid-sternum, there was a harsh-blowing systolic murmur, conducted as far as the edge of the sternum to the right, and beyond the anterior axillary line to the left. Over almost the entire sternal region there was a soft-blowing systolic murmur, quite different in character from that heard at the apex. It had the same tone throughout the whole sternal region, but it seemed to have two points of maximum intensity; to be more exact, it was loudest in the pulmonary region, exactly over the area of pulsation, from which point it waned in its intensity in every direction until near the lower end of the sternum, when it became louder, again culminating at the point where the left side of the sternum was joined by the sixth costal cartilage; but in this



FIG. 161.—Tracing from conus arteriosus in Case 38.

situation the murmur was not quite so loud as over the area of pulsation in the pulmonary region. The second sound was frequently reduplicated, and the later of the two second sounds, which could by auscultation be determined to be that due to the pulmonic cusps, was instantly followed by a short, sharp, high-pitched murmur perfectly soft in character. This murmur was extremely restricted in its distribution, being only heard over a small triangular area  $2\frac{1}{4}$  in. in vertical, and 2 in. in horizontal measurement, extending along the left border of the sternum from the lower border of the third costal cartilage to the upper border of the fifth. This murmur was perfectly soft in character, and was absolutely unlike the obstructive diastolic murmur which is found in mitral stenosis. It could not be due to aortic disease, of which there was no indication, and it could only, therefore, be a murmur of regurgitation from the pulmonary artery into the right ventricle, due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps. All these murmurs are shown in Fig. 162.

The other systems presented no symptom of disease, with the sole exception of a few crepitations at the bases of both lungs.

The diagnosis was cardiac dilatation, with mitral and tricuspid regurgitation, produced by the febrile affection, but it was considered probable that some obstruction at the mitral orifice might be insidiously progressing, although this was a mere supposition not based on any direct evidence. The crepitations at the base of the lungs were regarded as the expression of passive congestion from mitral incompetence; and the diastolic murmur was assumed to be one of pulmonary escape, in consequence of the strain on the artery from the high pressure within it.

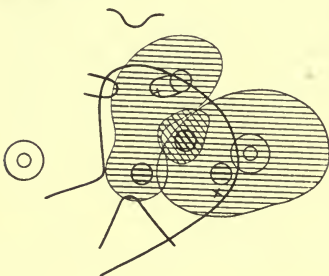


FIG. 162.—Systolic mitral and tricuspid murmurs with diastolic pulmonary murmur.

By means of salol and similar remedies the patient was relieved of her rheumatic symptoms, and the administration of iron with other tonics greatly improved the cardiac condition. The diastolic murmur disappeared and the lungs cleared up, but at the time of the patient's departure from the Royal Infirmary, on 17th July 1893, she still had the systolic murmurs, and the pulsation in the second intercostal space. She presented herself at the hospital on the 2nd March 1894, when the diastolic murmur was found to be still absent, but the systolic murmurs were present as before. The first sound in the mitral area, preceding the systolic murmur, was, however, loud and clanging in character, which seemed to bear out the view that a stenosis of the mitral orifice was gradually and insidiously developing.

In this case there was fortunately no opportunity of verifying the diagnosis, but in the following instance this was possible.

**CASE 39.—*Functional Pulmonary Incompetence.***—N. W., tablemaid, aged 16, was admitted to Ward 25 of the Royal Infirmary, 22nd July 1896, complaining of cough and breathlessness.

The patient's father died of disease of the aortic and pulmonary valves at the age of 45. His case is recorded on p. 576 (Case 37). Her mother, aged 44, was in good health. There had been three brothers and two sisters. One brother had died of scarlet fever. The others were quite well. The patient's previous health had never been at any time very strong, and she had suffered from acute rheumatism at the age of 8.

The patient, on admission, was found to be a delicate looking girl of slender build, and slight muscular development. The skin was pale in tint, excepting upon the cheeks, where there was a deep flush; the lips, ears, and nostrils were cyanotic. There was some fulness about the

ankles which pitted on pressure. The temperature on admission was  $101.4^{\circ}$ . Height 5 ft.  $1\frac{1}{2}$  in. Weight 6 st.  $2\frac{1}{2}$  lb.

The tongue was furred, but there was little apparent interference with the digestive processes. The liver was normal in size, and there were no morbid symptoms connected with the abdominal organs including the spleen.

The pulse rate was 128. The vessel was healthy, and the tension was low. The pulse was slightly irregular, and also somewhat unequal. On inspection of the neck, there were no morbid movements of the vessels, and the veins were not turgid. The apex beat was in the fifth intercostal space  $3\frac{1}{2}$  in. from mid-sternum. The upper part of the heart reached to the second left intercostal space. The right border at the fourth costal cartilage was  $2\frac{1}{2}$  in. from mid-sternum; the left border was 4 in. from mid-sternum. On auscultation two distinct murmurs could be determined. One of these—a high-pitched harsh murmur—had its point of maximum intensity 2 in. to the left of the mid-sternal line, at the level of the upper end of the ensiform cartilage; it was, therefore, situated almost half-way between the mitral and tricuspid areas. This murmur was propagated for a considerable distance over the præcordia. The other murmur, diastolic in time and extremely soft in character, was heard most distinctly in the third left intercostal space, its point of maximum intensity being  $1\frac{1}{2}$  in. from mid-sternum. This murmur immediately followed an extremely distinct second sound, which was very much louder to the left than to the right of the sternum. It could only be heard for a very short distance in every direction, but more particularly downwards towards the ensiform cartilage.

The respirations numbered 44 per minute, and were laboured. There was frequent cough attended by a considerable amount of frothy, serous expectoration. On examination of the lungs, it was found that the respiratory murmur was of a harsh vesicular description, with greatly prolonged expirations, attended by fine crepitations and sibilant rhonchi, but without any alteration in the vocal resonance. These features were presented by both lungs, but they were more pronounced on the right side. The urine was extremely scanty, but during the first four days of her residence in the ward it rose from 8 to 32 oz., under the influence of the remedies employed. It contained no abnormal constituents. The skin was bedewed with perspiration. The patient slept badly, but this was in great part the result of the harassing cough. With the exception of this insomnia, there was no alteration as regards the nervous system.

Consideration of the various features presented by the patient led to the conclusion that it was a case of organic mitral disease. The high-pitched, harsh systolic murmur situated midway between the mitral and tricuspid areas, conducted in every direction to a considerable distance, and propagated more particularly to the axilla and round to the neck and the scapula, could not but be sufficient evidence that there was mitral regurgitation, while the harshness and high pitch of the murmur seemed to prove most decidedly that the regurgitation took place through a constricted orifice. It was impossible to determine whether there was



also a tricuspid murmur, since the murmur already described was so loud as to obscure every other sound occurring during the systole of the heart. But from the implication of the peripheral veins, as shown by the œdema of the ankles, it seemed perfectly obvious that the right side of the heart was involved.

The chief difficulties presented by the case centred themselves, however, around the diastolic murmur. The mere localisation of this murmur in itself could not be held, in view of what has been already remarked, as evidence of any great value. It might have been either aortic or pulmonic in its origin, but the careful examination of the pulse showed that there was absolutely no tendency to the type of Corrigan, while the most diligent observation of the skin when reddened by friction failed to give any indication of a capillary pulse. The extremely soft character of the murmur, moreover, negatived any possibility of its being a diastolic murmur of a constricted mitral orifice.

The conclusion, therefore, was inevitable that the murmur was due to pulmonary incompetence; and two considerations rendered it almost certain that the incompetence was of the transient type produced by greatly increased pressure within the artery. There was, in the first place, not the slightest approach to any clubbing of the fingers, and, in the second place, the murmur varied greatly in its intensity from day to day, being at times almost inaudible and becoming very greatly intensified on the occurrence of any event throwing a greater strain upon the circulation.

The diagnosis arrived at, therefore, was mitral obstruction and incompetence with regurgitation at the pulmonary and tricuspid orifices, in consequence of dilatation from strain.

The patient was treated by means of hot poultices applied to the chest, with digitalis, ether, and ammonia internally.

The temperature fell, during the two days subsequent to her admission, to normal. The pulse rate and the respiration rate were also considerably diminished, while the functions of the kidneys were considerably improved.

The temperature again rose, however, after falling to the normal, but became practically steady within a fortnight of her admission. The condition of the lungs underwent great improvement, and the œdema disappeared from the ankles. During August and the first half of September this improvement continued, so that the patient was able to sit up and even move about the ward, but during the second half of September and the month of October a number of untoward symptoms returned, and in spite of every means adopted to meet them she died on the 21st October.

The result of the post-mortem examination was as follows:—

*External Appearances.*—The body was rather poorly developed, the surface was pale, with the exception of the face, which showed lividity. Rigidity was well marked. There was no dropsy.

*Thorax.*—The pericardial sac was normal. There were some scattered adhesions over both lungs.



*Heart.*—It was considerably enlarged, the left side being proportionately small. The right ventricle was much enlarged. The arterial valves were competent. *Aortic* =  $\cdot 9''$ ; *Pulmonary* =  $1\cdot 0''$ ; *Mitral* =  $\cdot 35''$ ; *Tricuspid* =  $1\cdot 3''$ . The mitral valve was extremely stenosed, forming a typical "button-hole" orifice. The segments were much thickened, adherent at their edges, partly calcified, and presenting a roughened margin on their auricular aspect. The chordæ tendineæ were thickened and shortened, but not to so great an extent as is often the case. The other valves were normal. The left ventricle was  $3\frac{1}{4}''$  in length, and its thickness of wall was  $\frac{3}{8}''$ . Part of the left ventricular wall was rather thinned. The left auricle was dilated, and its endocardium thickened and opaque. The right ventricle was much enlarged, and its wall greatly hypertrophied; its thickness at some places was  $\frac{3}{8}''$ . Its columnæ carneæ were thick and powerful, contrasting with those of the left ventricle, which were considerably atrophied. The right auricle was dilated, and there was a small thrombus in the appendix. The myocardium of the left ventricle was rather soft, but of normal appearance; that of the right ventricle was firm and rather pale. *Weight of Heart* =  $14\frac{1}{2}$  oz.

*Lungs.*—The right weighed 2 lb. It contained several large pulmonary infarcts; the largest was at the lower part of the upper lobe, and measured about 3" across. Those of larger size were of dull reddish colour, with some yellowish patches, and only a few areas of purple colour. The smaller were darker in colour. The lung tissue was in a condition of advanced chronic venous congestion, and was very tough. The left weighed 1 lb. 12 oz. and was in a similar condition; it also contained several infarcts, some of which were of large size and similar appearance to those in the other lung. There was some recent fibrinous exudation between the upper and lower lobes.

*Abdomen.*—The peritoneum was normal.

The *liver* weighed 3 lb. 6 oz. and showed typical chronic venous congestion of the "nutmeg" condition.

The *spleen* weighed 5 oz. It was of firm consistence; pulp of dark purple colour. Chronic venous congestion.

The *kidneys* each weighed 6 oz. and showed chronic venous congestion. No infarcts in spleen or kidneys.

The result of the post-mortem examination amply justified the diagnosis which had been made. The mitral obstruction was verified, and the absence of any structural changes at the pulmonary orifice proved that the diastolic murmur was caused in the manner suspected.

One other case of this kind may be mentioned, as presenting the same features connected with the pulmonary valve, but associated with different mitral appearances.

CASE 40.—*Functional Pulmonary Incompetence*.—A. L., aged 19, was admitted to Ward 25 of the Royal Infirmary in March 1897, suffering from chorea and debility. The patient's father, aged 42, and mother, aged 43, were in excellent health, as were three brothers and two sisters. Another sister had died of infantile diarrhoea at the age of seven months. Since the age of 7, the patient had always been somewhat delicate. At that age she had an attack of chorea, which had since then frequently recurred. During the last two or three years she had been getting gradually weaker. She had never suffered from acute rheumatism. The patient was well nourished, but presented a mingled appearance of cyanosis and anæmia. The face was pallid, with the exception of a dusky flush over the malar bones, the nose was thick, and the *alæ nasi* were bluish, as were also the lips and ears.

The teeth were bad, but there was no symptom of digestive disturbance. The abdominal viscera were normal in size, including the spleen. The hæmoglobin was 45 %, and the number of red blood corpuscles was 3,425,000. The radial artery was soft, elastic, and moderately full, with fair pressure. The pulsation was regular and equal, and the pulse rate was 80. There was no capillary pulse in the nails or over patches of redness produced by friction on the forehead and chest. There was no abnormal appearance connected with the neck or præcordia, but on palpation a very distinct thrill was perceptible at the apex of the heart, which was situated in the fifth intercostal space, 5 in. from the mid-sternal line. Careful palpation of this thrill showed that it coincided with the diastole, and died away before the systole. The apex beat was unattended by any palpable vibrations, but the closure of the semilunar cusps could be felt as a very distinct impact over a considerable area of the præcordia. On percussion, the right border of the heart was found, at the level of the fourth costal cartilage, to be  $1\frac{1}{2}$  in. from mid-sternum. At the same level the left border extended 5 in. from the mid-sternal line. In the fifth intercostal space the border of the heart was  $5\frac{1}{2}$  in. from mid-sternum. On auscultation in the aortic area, both sounds were clear and distinct. In the mitral area there was a soft-blowing systolic murmur propagated across for some distance towards the sternum, and for a considerably greater distance towards the axilla. There was also a harsh murmur commencing with the second sound, and prolonged over a considerable portion of the diastolic phase, but ceasing some time before the first sound. In the pulmonary area the first sound was pure, but the second sound was greatly accentuated, and was immediately followed by a short soft murmur rather high-pitched in tone, and entirely different in all its characters from the diastolic murmur heard in the mitral area. The maximum intensity of this murmur was at the sternal end of the third left intercostal space. It was conducted about half-way up the manubrium sterni, but there was no trace of murmur over the carotid arteries. In the tricuspid area the sounds were unaltered.

There were some crepitations at the bases of both lungs, but otherwise the respiratory system presented no symptoms requiring notice. The

only other point in the case which requires any remark is in regard to the nervous system. The patient presented characteristic choreic movements, mostly confined to the left side, but as this hemichorea has no immediate connection with the subject under discussion, it is unnecessary to dwell upon it.

Under treatment with arsenic and digitalis the patient rapidly recovered and was discharged.

The case which has just been narrated furnishes a most typical example of relative pulmonary incompetence. That the soft-blowing diastolic murmur which immediately followed the accentuated second pulmonary sound was due to a reflux at that valve, could not for a moment be doubted. It was certainly not the diastolic obstructive mitral murmur, since the characteristics of the two were so essentially different. It was impossible further to conceive of it as being due to any aortic lesion, since there was no evidence in the arteries or capillaries of any disturbance at the aortic cusps.

One important fact has impressed me very strongly on considering cases such as these. This murmur of high pressure in the pulmonary artery does not seem to be usually associated with tricuspid incompetence, and it is probable that the existence of free regurgitation at the right auriculo-ventricular orifice is able to lessen the pulmonary pressure to such a degree that the murmur cannot be produced.

## PULMONARY OBSTRUCTION AND REGURGITATION.

A discussion of the pathological and clinical features of combined obstruction and incompetence at the pulmonary orifice would be quite out of place, seeing that the results are simply those of a double lesion in which one or the other element preponderates.

## CHAPTER XIII.

### AFFECTIONS OF THE TRICUSPID ORIFICE.

LESIONS affecting the right auriculo-ventricular orifice present a remarkable contrast in respect of frequency. Tricuspid obstruction is one of the rarest valvular lesions; regurgitation at this orifice is beyond compare the most common. Statistics do not bring this fact out with such clearness as they should. The reason, however, is not far to seek. Incompetence of the tricuspid valve does not in itself seriously impair the general course of the circulation, and it is therefore often found amongst those who, although under treatment for various affections, have no cardiac symptoms. It accordingly escapes observation unless especially sought for.

### TRICUSPID OBSTRUCTION.

The anatomical features of this lesion have been known since the time of Morgagni, who described a case in which the lesion was discovered after death in association with a similar affection of the mitral orifice. Corvisart carefully recorded the symptoms observed during life and the lesions found after death in a case of combined tricuspid and mitral obstruction, and he further mentions a pathological specimen obtained from a patient with similar morbid changes, adding that he could have given several other instances of the same nature. Horn shortly afterwards recorded a case in the person of a woman, aged 25, whose heart was observed, post-mortem, to have obstruction of both right and left auriculo-ventricular orifices caused by valvular changes.



Burns, a year later, gave a full and clear account of the symptomatology and pathology of similar double lesions as they occurred in a young woman, 19 years of age. Bertin described an instance of obstruction of both right and left auriculo-ventricular orifices, and another in which the tricuspid orifice alone was involved. He further cites a case which had been recorded by Corvisart, Leroux, and Boyer. Bouillaud incorporated in his work the description of the case of the general officer, which was recorded by Corvisart, Leroux, and Boyer, and referred to by Bertin. The lesions found in this case after death were somewhat singular. The tricuspid valves were so united as to close the orifice with a diaphragm penetrated by three apertures, two of which opened from the auricle into the ventricle, while the third ended in the left ventricle. In this case the left side of the heart was otherwise in a normal condition.

Since the date of these observations many cases of tricuspid obstruction have been recorded, but comparatively few of them were recognised during life. Before the date of Laennec's great discovery, Kreysig attempted to formulate rules by which tricuspid obstruction might be diagnosed, and, after the introduction of auscultation, Hope drew up a clear statement of the local symptoms and physical signs which might be expected to occur. In most of our systematic treatises, whether devoted specially to the diseases of the heart or embracing the wider sphere of general medicine, the subject of tricuspid stenosis is dismissed in a few theoretical sentences. This is the case not only with the majority of our English works, but with those also by transatlantic and continental authors. A small number of writers, however, have laid before us some positive observations on this form of valvular disease, from which useful general principles as to diagnosis may be drawn.

The extremely interesting case which Gairdner placed on record in 1862, and which he has since fully described, was the first instance of a diagnosis of obstruction of the right auriculo-ventricular orifice made during life and verified by post-mortem examination. Haldane showed at a meeting of the Edinburgh Medico-Chirurgical Society, in 1864, two specimens of obstruction of the tricuspid orifice, in both of which mitral obstruction was

also present. In one of these the patient during life presented no physical signs which could lead to a diagnosis of the tricuspid lesion, and in this respect it closely resembles a case which Philip has narrated. In the other patient the lesions were recognised during life by the existence of a presystolic murmur heard at the apex, and another of similar rhythm at the lower end of the sternum, between which points of maximum intensity the murmur was not so loud. Duroziez described ten cases, two in men and eight in women. Walshe would appear in his long experience only to have met with a single case in which this lesion probably existed, but as there was no post-mortem examination he in consequence speaks very guardedly on the subject of diagnosis. In the exhaustive work of Hayden there is an account of a large number of cases recorded in the United Kingdom and America, including three observed by the author, two of which were recognised during life. In every one of these instances there was a lesion of the mitral as well as of the tricuspid orifice. Bedford Fenwick, when showing two cases of tricuspid obstruction at the Pathological Society, analysed the facts presented by 46 instances on record. The most important conclusions to which he was led are, that it is more common in women than in men in the proportion of 41 : 5, that it is almost always associated with mitral obstruction, and that there is a history of rheumatism in about 50 per cent. of the cases observed. By the researches of Leudet, 117 instances of acquired tricuspid obstruction have been collected from medical literature, including those embraced by the previous writers already mentioned. In 114 of these cases there were post-mortem examinations, allowing a wide field for observation. Of these cases, 86 occurred in women, 22 in men, and in 6 the sex was not mentioned. Leudet's results with regard to the implication of the different orifices in tricuspid obstruction are as follows:—

Obstruction of tricuspid alone . . . . .	11
„ tricuspid and mitral . . . . .	78
„ tricuspid and pulmonary . . . . .	3
„ tricuspid, mitral, and aortic . . . . .	21
„ tricuspid, mitral, and pulmonary . . . . .	1

Herrick has brought the tale of recorded cases down to the present date. He finds that since the publication of Leudet's work in 1888, 40 cases with autopsies have been narrated, 28 of which occurred in women and 10 in men, while in 2 cases the sex was not noted. The valvular lesions present in those 40 cases were—

Obstruction of tricuspid alone	.	.	.	.	1
„ tricuspid and mitral	.	.	.	.	18
„ tricuspid and pulmonary	.	.	.	.	0
„ tricuspid, mitral, and aortic	.	.	.	.	18
„ tricuspid, mitral, and pulmonary	.	.	.	.	1
„ tricuspid, mitral, aortic, and pulmonary	.	.	.	.	1
„ tricuspid and aortic	.	.	.	.	1
					<hr/>
					40

Summing the results furnished by Leudet and Herrick there have therefore been 154 cases more or less thoroughly recorded, of which 114 occurred in women and 32 in men, while in 8 the sex was not mentioned. The results of post-mortem examination in those 154 cases showed the following associations of valvular lesions:—

Obstruction of tricuspid alone	.	.	.	.	12
„ tricuspid and mitral	.	.	.	.	96
„ tricuspid and pulmonary	.	.	.	.	3
„ tricuspid, mitral, and aortic	.	.	.	.	39
„ tricuspid, mitral, aortic, and pulmonary	.	.	.	.	1
„ tricuspid, mitral, and pulmonary	.	.	.	.	2
„ tricuspid and aortic	.	.	.	.	1
					<hr/>
					154

ETIOLOGY.—The influence of sex has already been shown in presenting the results of previous observers. In 146 cases verified by post-mortem examination, in which the sex is mentioned, 114 occurred in women and 32 in men; that is, in the proportion of 3.5:1. A considerable number of cases of tricuspid obstruction are of congenital origin, and the proportion is believed by Peacock and Rosenstein to be so great as to constitute the majority of cases of the affection. The views, however, of these authors are not borne out by the observations

of the majority of authors, and in the larger proportion of cases the affection undeniably takes its origin after birth.



FIG. 163.—Tricuspid obstruction.

Rheumatism and chorea give rise to a considerable number of cases. The painstaking researches of Herrick show that in



the 154 cases which he has analysed, rheumatism was present in 51, doubtful rheumatism in 4, chorea in 2, while there was no history of rheumatism in 28, and in 69 no facts bearing on etiology were noted. It is probable that some of the acute specific diseases are responsible for cases of tricuspid obstruction, as in an instance recorded below. When all sources are investigated, there remain, nevertheless, many without any obvious origin; that chronic valvulitis produced by severe exertion may cause it, appears to me at least reasonable.

**MORBID ANATOMY.**—The structural alterations which take place in tricuspid obstruction are in the main similar to those found in obstruction of the left auriculo-ventricular orifice. The most frequent alteration observed is the union of the cusps so that they form a funnel-shaped structure surrounding the orifice. Sometimes, but rarely, the union of the cusps produces such a degree of obstruction as scarcely to admit the point of the little finger. Lesions of this kind have been more particularly described by Duroziez, but they are far from common. In other cases, in addition to a certain amount of union of the cusps, there is some contraction with rigidity, and there may even be some deposition of inorganic salts. There may be vegetations, either of recent origin or of older standing. In some instances, as in that figured in the accompanying illustration (Fig. 166, p. 597), the lesions consist in fine transparent granulations, in other cases these granulations have undergone the fibrinous changes already described in the case of the mitral orifice.

The secondary results upon the heart have never been very clearly studied, since, with few exceptions, the affection has always been accompanied by a similar lesion of the mitral orifice. It would naturally be expected that some hypertrophy of the left auricle would follow the development of the lesion, and this, as a matter of fact, is the case. It has, however, in addition, been accompanied by a certain amount of dilatation, as will be seen to have been the case in some of the instances narrated below. The illustrations (Figs. 164, 165, and 166) show some of these effects. They are from Case 42, in which (p. 604) the full details can be read. They show enormous dilatation and hypertrophy of the right auricle and ventricle.

These results are, however, almost certainly produced by the great constriction of the mitral orifice, and throw no light



FIG. 164.—Great dilatation and hypertrophy of the right auricle and ventricle from combined mitral and tricuspid obstruction and incompetence. The heart is seen from the front.

upon the consequences of tricuspid obstruction. When the affection has been of congenital origin it has been accompanied, as might be expected, by some other malformations,

such as pulmonary obstruction, patent foramen ovale, or patent



FIG. 165.—Great dilatation and hypertrophy of the right auricle and ventricle from combined mitral and tricuspid obstruction and incompetence. The heart is seen in profile from the right.

ductus arteriosus. Upon the circulation the effect of the

disease is to bring about a considerable degree of venous stasis, and this shows itself by characteristic clinical features.

**SYMPTOMS.**—On account of the almost invariable association of tricuspid obstruction with some other cardiac lesion, it is difficult to be certain how much of the symptomatology is due to the right-sided lesion; many symptoms which present themselves are due without doubt to secondary changes in the right side of the heart. There may be absolutely no evidence leading to suspicion of the tricuspid lesion. In cases, nevertheless, where a very considerable degree of obstruction of the tricuspid orifice has existed, there is some dyspnoea, exaggerated on exertion, but there is a greater degree of cyanosis than of breathlessness, and it is associated with chilliness of the extremities and great susceptibility to cold. As Foster pointed out, there is more tendency to stasis of the systemic than of the pulmonic veins.

On observing the general appearance of any patient, therefore, suffering from this disease, the complexion is usually seen to be dusky, with dark lips, nostrils, and ears. There is commonly some oedema about the ankles. The urinary secretion is scanty and high coloured, often containing albumin. The cerebral faculties are often impaired in one direction or another, and, on physical examination, the liver and spleen are somewhat enlarged, while ascites may be present. It is highly probable that many of these symptoms are not directly produced by the tricuspid obstruction, but they must in many instances be exaggerated by it.

The physical signs are sometimes extremely well marked. On examining the veins of the neck they are sometimes seen to be turgid and motionless, in other instances they show well-marked venous pulsation. The investigations of Mackenzie, already discussed, have thrown much light on this subject.

The radial artery may present no alteration. It may be of average fulness and pressure, and show no alteration in rhythm or rate; but it may, on the other hand, be empty, compressible, irregular, and frequent.

Palpation has in some instances revealed a well-marked purring thrill. The area over which this thrill is felt is usually wide, and sometimes it has been possible to make out



two points of maximum intensity, one at the apex of the heart at the fifth intercostal space, the other just outside the left edge of the sternum. But in other cases the thrill has its point of maximum intensity somewhere between the mitral and tricuspid areas, as was the case in one of the instances narrated below.



FIG. 166.—Transverse section through the heart in combined mitral and tricuspid obstruction and incompetence. The button-hole mitral orifice is fused with the chordæ tendineæ; the triangular tricuspid orifice, partly hidden by the papillary muscles, has vegetations on its margins; the right ventricle is dilated and its walls greatly hypertrophied.

No constant condition is found on percussion, but in most instances the area of cardiac dulness is increased transversely, both borders of the heart being situated too far out. In some instances there can be no doubt that the right auricle is considerably dilated.

On auscultation the characteristic phenomenon is a pre-systolic or diastolic murmur, heard with its greatest intensity

at the junction of the fifth and sixth ribs with the left side of the sternum. Occasionally the murmur begins with the second sound and fills up the entire interval between it and the first sound with some presystolic reinforcement. It must be admitted, however, that in many cases no murmur has ever been detected during life, and, therefore, the lesion occurs as a post-mortem surprise.

DIAGNOSIS.—The recognition of tricuspid obstruction depends almost entirely on the presence of positive physical signs, seeing that the symptoms produced by interference with the circulation are similar to those arising from the secondary results of mitral disease, which has been seen to be almost invariably associated with tricuspid obstruction. The appearances in the veins of the neck to which reference has been made are precisely those found in secondary implication of the right ventricle in the course of mitral disease, and the same may be said of the enlargement of the area of cardiac dulness to the right. It is otherwise, however, with the evidence afforded by auscultation. The presence of the presystolic or diastolic murmur with its maximum intensity in the tricuspid area must be allowed to be pathognomonic of obstruction of the right auriculo-ventricular orifice. In certain cases a murmur of one or other of these rhythms is accompanied by a murmur of mitral obstruction, in which case the murmurs have separate points of maximum intensity, one in the tricuspid, the other in the mitral area. In other cases, as in one (No. 44, p. 607) described below, only one murmur can be determined, having a point of maximum intensity intermediate between the mitral and tricuspid areas. It must be admitted in such instances that the diagnosis is a matter for discussion, and each instance of this nature must be judged on its own merits.

In a large proportion of cases of tricuspid obstruction the lesion appears to elude observation, probably in consequence of the absence of the murmur. Just as murmurs of mitral obstruction are variable, it is probable that similar tricuspid murmurs have a tendency to appear and disappear.

Hayden, in his remarks on tricuspid obstruction previously referred to, mentions a case in which he had diagnosed aortic

and mitral obstruction. These lesions were found on post-mortem examination, but there was also tricuspid obstruction which had not been diagnosed. The author reproduces some observations which he made at the Dublin Pathological Society, which may well be quoted here. He remarked that "diagnostically the case is of considerable interest. It is perfectly novel to me, and, with the light it affords, I should have no difficulty in diagnosing, in a similar case, the existence of constriction of the two auriculo-ventricular openings. The point on which the diagnosis turns is this, that whereas the murmur of mitral constriction is always at the apex of the heart, and, in the great majority of cases, strictly limited to the area of the mitral opening, in this case a murmur of the same rhythm was audible to the left of the sternum. Between these two points there was a portion of the chest over which no murmur was distinctly audible." Profiting by the experience thus gained, the author just quoted was able, as mentioned previously, to diagnose during life, and verify after death, two later cases of tricuspid stenosis.

Balfour speaks of a boy, who, he says, "had a presystolic murmur so loud and rough that I have selected it as a measure of the extent to which such murmurs could be propagated. In mapping out the propagation of his murmur, I found it to extend so much further to the right than usual that the thought struck me, is it possible that we can have in this case not only a mitral but also a tricuspid stenosis? But I dismissed the idea as in the highest degree improbable, and referred the great propagation to the loudness and roughness of the mitral murmur. The result shows that in this I was mistaken, though unquestionably there were no other symptoms present but the excessive propagation of the murmur which could countenance the former idea." In this case considerable obstruction of both mitral and tricuspid orifices was found on post-mortem examination. The result of this case was such as to lead Balfour to diagnose in a similar case combined mitral and tricuspid obstruction; but as down to the time of the publication of the second edition of his work the patient was alive, the diagnosis was not absolutely confirmed.

Mackenzie has suggested that, in the frequent absence of a

presystolic murmur, obstruction of the right auriculo-ventricular orifice may be suspected when pulsation of the liver exhibits an auricular type—that is to say, when the principal impulse takes place in advance of the ventricular systole. The explanation which he gives is that in consequence of the obstruction the hypertrophied auricle sends blood backwards as well as forwards, and this produces the auricular liver pulse. His conclusions were based on the examination of seven cases of tricuspid obstruction, in five of which the condition was confirmed by post-mortem examination.

PROGNOSIS.—A forecast of the future must of necessity be difficult in cases of tricuspid obstruction, since the affection is almost invariably complicated by the presence of other lesions, and the prognosis can only be the result of careful consideration in regard to each individual case. Duroziez has, nevertheless, boldly attempted to formulate rules for guidance in respect of prognosis. He has pointed out that, while the average age of death is thirty-two years in the case of such obstruction as will not admit the point of the finger, the duration of life reaches forty-two years in those cases where the orifice allows the passage of two fingers. This must be allowed to be a matter of small concern to the practical physician, from the obvious fact that we have no means even of guessing the size of the orifice.

TREATMENT.—Tricuspid obstruction if at all considerable diminishes the blood supply to the lungs, and must in this way be attended by persistent dyspnœa. It also causes retardation of the return of blood from the systemic veins and thus leads also to general venous stasis. The indications for special treatment are, therefore, to get rid of any local troubles which may tend to increase the interference with the functions of those parts more particularly implicated, while carrying out the general lines of treatment applicable to valvular disease.

CASE 41. *Mitral and Tricuspid Obstruction and Incompetence.*—Isabella F., æt. 31, domestic servant, was admitted on 1st April 1892 to Ward 25, then under my care, suffering from right-sided hemiplegia, with aphasia. Her father died at the age of 72, of bronchitis. Her mother, 65 years old, always enjoyed good health. She had three brothers and four sisters, who had always been healthy. Her social surroundings were satisfactory. Ten years before she had suffered from



a severe attack of acute rheumatism, since which time her health had not been so good as formerly. The present attack began about nine months before admission. After undergoing some mental troubles the patient suddenly found that she had lost the power of moving the right arm, and, on attempting to inform some one of the circumstance, discovered that she was unable to do so. She was told by another servant that her face was drawn to the left side. It was deemed advisable that she should go to bed, and after betaking herself thither the patient gradually lost the power of moving the right leg. After the lapse of a few weeks she partly regained the use of the leg and arm, and to a less extent that of the face, but speech was only restored in a very slight degree.

On examination it was found that the alimentary and hæmopoietic systems were in no way affected. Inspection showed a faint double reflux—auricular- and ventricular-systolic in rhythm respectively—in the veins of the neck. The apex beat was very distinct on account of the thinness of the patient; it was situated in the sixth intercostal space,  $4\frac{1}{4}$  in. from the middle line. There was some pulsation in the epigastrium. On palpation it was noticed that there was at times a faint diffuse thrill immediately preceding the cardiac impulse. It was felt to be most

distinct at the apex, but could be traced almost as far as the xiphoid cartilage. The radial pulse was of moderate fullness and pressure; its rate was usually about 80, and its rhythm regular. The cardiac dullness began above at the lower edge of the third left costal cartilage. The right border of the heart was  $1\frac{1}{2}$ , and the left  $4\frac{1}{2}$  in. from the mid-sternal line. At the apex there was a distinct, but not loud, murmur of presystolic rhythm and rough character, which was succeeded by a soft blowing systolic murmur, followed in its turn by a double second sound. When these murmurs were traced, it was found that the one of presystolic time

could only be heard for about an inch to the left of the apex, but that it was propagated as far as the ensiform cartilage, slightly diminishing in loudness for a certain distance, and, after that point had been passed, increasing in intensity and altering in character as the lower end of the sternum was approached. The systolic murmur was conducted as far as the axilla on the one hand, and to the xiphisternum on the other, but, as in the case of the presystolic murmur, there was a decrease of intensity on auscultating across the space from the apex to the sternum, followed by a gain in loudness when the ensiform process was approached. There were, in short, presystolic and systolic murmurs having maximum intensity both in the mitral and tricuspid areas. At the base of the heart the first sound was

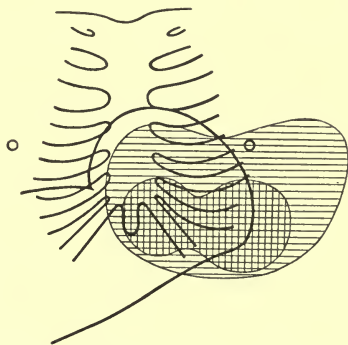


FIG. 167.—Presystolic and systolic mitral and tricuspid murmurs in Case 41.

very feeble, and the second sound reduplicated, the pulmonary part of it being at once louder and later than the aortic. The position and extent of the abnormal sounds are shown in Fig. 167.

The respiratory system had but moderate deviations from the standard of healthy conditions, there being a little muffling of the percussion sound, a slight increase in the roughness of the respiratory murmur, and a few scattered crepitations over the whole of the thorax. Neither the integumentary nor urinary functions were in any way affected.

With regard to the nervous system, it was found that there was no alteration in sensibility, ordinary or special. The right leg and arm were able to perform most movements, but in a weak and sluggish manner, and the toes and fingers moved very imperfectly. The mouth was drawn slightly to the left side when the patient smiled, and the tongue tended towards the right on protrusion. The muscles of the right leg and arm were wasted, and the face on that side was expressionless. The right half of the entire muscular system showed diminished reaction both to galvanism and faradism. The temperature of the paralysed side was lower than that of the other. The plantar and other superficial reflexes were exaggerated on the right side of the body, and on that side there was great increase in the knee jerk, and a marked ankle clonus, as well as an exaggeration of the elbow jerk, and a distinct wrist clonus. Even from the date of the paralytic seizure, the patient had been able to understand everything said in her presence, and to read with perfect ease; she had, in point of fact, spent most of her time in reading. At the time of the attack she was unable in any way to express her ideas, but she had gradually regained the power of saying a few words, and had taught herself to write answers to questions with her left hand.

The diagnosis arrived at in this very interesting case was obstruction of both auriculo-ventricular orifices, with incompetence of their respective valves, and embolism of a branch of the left middle cerebral artery, involving the motor tracts corresponding to the centres for the leg, arm, face, and speech.

After some gradual improvement in the general condition, and in the state of the nervous system, the patient's circulation showed symptoms of failure. The lungs became œdematous, and some anasarca of the lower extremities followed. Under appropriate treatment these symptoms, especially as regards the limbs, lessened to some extent, but on the 27th April death occurred suddenly from cardiac failure.

The post-mortem examination was performed on the day after death by Dr. William Russell. The following is the description of the morbid anatomy from the books of the pathological department:—

*External Appearances.*—Body spare. Rigor present. Face, neck, and upper part of thorax livid. No anasarca.

*Thorax.*—There were a few ounces of fluid in each pleural cavity. No adhesions were found. There were about two ounces of clear fluid in the pericardium, and a patch of pericardial adhesion over the base of the right ventricle.

*Heart.*—There was a large and tough pinkish clot in the left ventricle,

which extended into the aorta. There was a similar clot in the right ventricle extending into the pulmonary artery, accompanied by a little post-mortem clot in the same chamber. Diameters of orifices: aortic, .97; pulmonary, 1.2; mitral, 1.1; tricuspid, 1 in. The aorta was normal, with the cusps slightly thickened, but not at all shrunken. Both cusps of the mitral valve were thickened and fibroid, as also were the chordæ tendineæ and apices of papillary muscles. From the anterior cusp there was suspended an irregular elongated mass of vegetation, which evidently hung free in the blood stream, and the tendinous cord with which it was united showed a small warty mass of vegetation. The auricular aspect of the posterior segment, and the auricular endocardium continuous with it, were occupied by an extensive warty growth, covering an area of about  $1\frac{1}{2}$  in. square. The left ventricle was dilated, and its muscle soft, pale, cloudy, and somewhat fatty. The left auricle was dilated and slightly thickened. The pulmonary artery was somewhat dilated. The right ventricle was elongated, but not much dilated; its wall was not hypertrophied, and its muscle was in the same condition as that of the left ventricle. The tricuspid valves were so united as to form a ring which admitted two fingers, constituting stenosis. The ring was adherent to the endocardium three-quarters of an inch below the pulmonary valves. The right auricle was much dilated, and the foramen ovale was closed. The heart weighed 1 lb. 6 oz. The appearance of the heart is shown in Fig. 163, p. 592.

*Left Lung* weighed 1 lb. 4 oz. It was emphysematous, anæmic, and œdematous.

*Right Lung* weighed 1 lb. 15 oz. It was emphysematous, and showed slight chronic venous congestion, with œdema.

*Head.*—Brain weighed 3 lb. 2 oz., and was extremely pale and bloodless. A vertical section through the ascending parietal convolution, revealed in the parietal fasciculus above the island of Reil an area of brownish pigmentation, with a degree of softening which was no doubt due to embolism. The other parts of the brain and the upper part of the cord were normal to the naked eye. No embolism in large vessels.

*Abdomen.*—Liver weighed 4 lb. 1 oz., was congested, and the margins of the lobules fatty. The gall bladder was œdematous, and contained a little greenish bile. Spleen weighed 1 lb. 2 oz. The capsule was tight and thickened. On section the organ was firm, the fibrous tissue was very prominent and thickened, and the Malpighian bodies were somewhat enlarged. Left kidney weighed  $6\frac{1}{2}$  oz. This was very anæmic and mottled with a diffuse gray colour. The capsule was adherent, and the markings in cortex were obscured and broken up. Right kidney weighed  $6\frac{1}{2}$  oz.; in the same condition as its fellow.

In this case the diagnosis of the double cardiac lesion which we were led to form in consequence of the distribution of the murmurs, was justified by the result of the post-mortem examination.



CASE 42. *Obstruction and Regurgitation of Mitral and Tricuspid Orifices, with Aortic Obstruction and Pulmonary Incompetence.*—J. L., aged 25, domestic servant, was admitted to the Royal Infirmary on the evening of 9th December 1897 on account of dropsy and breathlessness. Her condition on admission was serious, and precluded the possibility of obtaining exact information as regards the history of the illness as well as that of her family. Unfortunately no opportunity was allowed me of seeing the patient during life, but my resident physician, Dr. J. W. Simpson, has kindly furnished me with notes of the case.

There was a slight degree of cyanosis as evidenced by the dusky tint of complexion, the dark purple of the lips, and the lividity of the ears and nostrils, as well as the nails of the hands and feet. There was great œdema of the legs, of the arms, and of the back, and extensive ascites was also present. The tongue was dusky in hue, with a slight fur upon the surface. The liver extended from the fourth rib to 3 in. below the costal margin, or  $5\frac{1}{2}$  in. in total extent in the line of the mammilla. The radial arteries were healthy, the pulse pressure low, and the pulsation irregular; its rate was 80 soon after admission. On examination of the neck and præcordia, there was great distension of the jugular veins, and a general heaving impulse over the entire præcordia. On placing the hand over the chest, a faint presystolic thrill could be made out over the mitral area. The area of cardiac dulness appeared to be enlarged both to the right and to the left, but on account of the condition in which the patient was, no attempt was made to define the borders accurately. On auscultation both a presystolic and a diastolic murmur were heard; the presystolic murmur was somewhat widely diffused. The systolic murmur was heard over the entire præcordia.

The respirations were 36 per minute soon after admission, and there was evidence at the bases of hyperæmia, if not œdema, the percussion sound being somewhat dulled and crepitations being abundant. The renal secretion contained bile and albumin. In spite of every effort to relieve the patient, she speedily sank into a comatose condition, and died early on the morning following her admission.

The post-mortem examination, which was performed by Dr. R. A. Fleming, revealed an interesting condition of matters. There was well-marked lividity and some rigidity, more especially of the lower limbs, the arms being almost flaccid. Marked œdema was present both of arms and legs. The body was well nourished and the muscularity fair.

The pericardial sac contained 8 oz. of a slightly blood-stained fluid. The heart weighed 1 lb. 5 oz. before being opened. Both auricles were greatly enlarged; the right measured 5 in. longitudinally, and  $3\frac{1}{2}$  in. transversely; the left auricle was considerably dilated and somewhat hypertrophied. The right ventricle was enormously dilated and hypertrophied, its cavity was  $3\frac{3}{4}$  in. long, and its walls from  $\frac{3}{8}$  to  $\frac{5}{8}$  in. thick; the left ventricle was smaller than usual, its cavity being  $2\frac{3}{4}$  in. long, and the thickness of the wall  $\frac{1}{2}$  to  $\frac{3}{4}$  in.

The aortic cusps were competent, the pulmonary were markedly incompetent, and the orifice greatly dilated. The mitral orifice was



greatly contracted, the cusps, chordae tendineae, and tips of the papillary muscles being fused so as to form a thick partition between the auricle and the ventricle, perforated by a button-hole mitral orifice showing a cone diameter of  $\cdot 3$  in. The muscoli papillares were greatly hypertrophied. The tricuspid orifice was also obstructed, the three cusps being fused at their extremities, and the chordae tendineae being shortened and thickened; the papillary muscles and fleshy columns were much increased. Upon the three margins of the triangular orifice thus produced were some recent vegetations. The orifice showed a cone diameter of  $\cdot 5$  in., the aortic orifice had a cone diameter of  $\cdot 7$  in., and the cusps were the seat of numerous vegetations. The pulmonary orifice measured  $\cdot 9$  in. by the cone; its cusps were healthy.

The coronary arteries were tortuous and thickened. The right ventricle formed the anatomical apex of the heart.

The right lung weighed 22 oz. and the left 15 oz.; both showed marked hypostatic congestion and œdema. There was a small patch of collapse in the left lung near the apex of the upper lobe, and in the right lung, near the lower part of the upper lobe, there was a nodule about the size of a walnut which contained caseous material, which had undergone some calcareous degeneration. There was one small recent pulmonary hæmorrhage in the lower lobe of the left lung.

The liver weighed 3 lb. 6 oz., and showed very marked perihepatitis both of old and recent development. It showed well-marked cyanotic atrophy, but there was no evidence to the naked eye of any connective tissue increase in the portal spaces. On microscopic examination some commencing perivascular cirrhosis was observed.

The spleen weighed 7 oz. and showed marked perisplenitis along with chronic venous congestion.

Each kidney was 6 oz. in weight, and each showed chronic venous stasis.

It was most unfortunate that this case, so interesting in its morbid anatomy, did not come under more extensive clinical observation. It must be added to the category of those only realised on the table of the pathologist.

**CASE 43. *Tricuspid and Mitral Obstruction and Incompetence.***—Mrs. W., aged 30, hawker, was admitted to Ward 27 of the Royal Infirmary, 26th February 1898; on account of cough and breathlessness.

Her mother had died suddenly at the age of 49 from heart disease; her father died when she was very young, and she never knew the cause of his death. The family consisted of one brother and three sisters, who were perfectly well. She had been married for eleven years, but had never had any children, and for one year she had been a widow. Her general surroundings had not been particularly good, as her house was damp and draughty, while her occupation exposed her to adverse weather influences. There were no previous illnesses of much importance; in

fact, with the exception of measles, and catarrhs, she had never been ill; in particular, there had never been any rheumatic symptoms. For the three years previous to admission she had been troubled with winter cough, but during the warm weather it always disappeared. The patient was found to be a plump and healthy-looking woman, with ruddy cheeks, and clear complexion. The merest trace of cyanosis was present at the time of her admission, but speedily passed away. Her height was 5 ft.  $1\frac{1}{2}$  in., and her weight 6 st. 6 lb.

The alimentary system showed absolutely no abnormal symptoms, and the abdominal viscera were of normal size, the liver in particular extended from the upper border of the fourth rib to the costal margin, a distance of 5 in. The spleen was not enlarged, and the lymphatic glands throughout the body were normal.

For about two years before admission the patient had suffered from occasional attacks of pain in the præcordia. This was sometimes dull in character, but at other times was extremely sharp and caused her to hold her breath. There had been breathlessness on exertion for about the same period of time, and occasionally it seemed to take the form of cardiac asthma, which prevented her lying down during the night. There had latterly been some feelings of faintness, but never any loss of consciousness. Palpitation was present on exertion.

Diffuse pulsation was visible in the fourth, fifth, and sixth left intercostal spaces, as well as in the epigastrium and episternal notch. There was some pulsation of the veins on the right side of the neck; the impulse preceded the apex beat, and therefore was auricular. On palpation

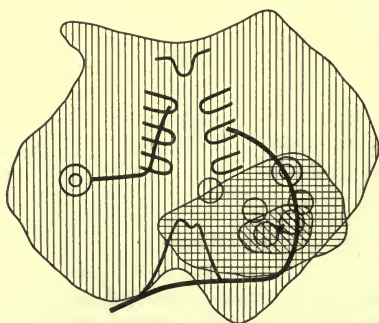


FIG. 168.—Murmurs of tricuspid and mitral obstruction and incompetence in Case 43.

the diffuse impulse was found to have its maximum intensity in the fifth intercostal space,  $3\frac{1}{2}$  in. from mid-sternum, but it was widely conducted. The impulse was accompanied by a distinct presystolic thrill, running up to the apex beat, and there was also an impulse corresponding to the second sound, occasionally followed by a diastolic thrill. On percussion the upper border of the heart was found to be at the inferior edge of the third left costal cartilage, and the cardiac dullness extended  $1\frac{1}{2}$  in. to

right, and 4 in. to left of mid-sternum at the level of the fourth costal cartilage. On auscultation over almost any part of the præcordia a more or less definite presystolic murmur could be heard, and on following this murmur over the præcordia it was found to have two points of maximum intensity. One of these, at which it was most distinct, was within the conventional tricuspid area at a point corresponding to the sternal end of

the fifth left costal cartilage. The other point of maximum intensity was within the mitral region, its loudest point being just inside of the apex beat. The presystolic murmur was much louder in the tricuspid region than in the mitral, and the two murmurs were distinctly different in tone. In the mitral area it was followed by a soft blowing systolic murmur, which practically replaced the first sound, and was succeeded by an accentuation of the second sound, from which a short rough diastolic murmur tailed away. In the tricuspid area the presystolic murmur was followed by a distinct first sound, which, however, passed into a faint blowing systolic murmur. The second sound in this area seemed to be of normal intensity, and there was no diastolic murmur. In the aortic area the sounds in no way differed from those of health, and in the pulmonary area there was a distinct accentuation of the second sound. The area of conduction of the presystolic murmur was extremely wide, measuring 12 in. vertically and 14 in. horizontally. The diastolic murmur was limited to a very small area, around the apex beat, measuring 2 in. transversely, and  $1\frac{1}{2}$  in. vertically. The systolic murmur was somewhat more widely conducted, and occupied an area of  $7\frac{1}{4}$  in. by  $4\frac{1}{2}$  in. The arteries throughout the body showed evidence of slight thickening. The vessels were full, and the pressure within moderate limits. The rate of the pulse was 68. It was almost regular in time and equal in size. Each individual pulse wave was somewhat small.

The respiratory system gave evidence of some subacute bronchitis on admission. This speedily passed away. There were no symptoms of disturbance connected with any other system in the body.

After two or three weeks' tonic treatment the patient was able to leave hospital. The physical signs remained as above described.

There could be absolutely no doubt that in this case there was obstruction and regurgitation at the tricuspid, as well as at the mitral orifice.

CASE 44. Marion R., æt. 21, machinist, presented herself as an out-patient on the 5th November 1892.

Her father, æt. 62, had for some time suffered from asthma; her mother, æt. 58, was in good health. Two brothers and two sisters were in perfect health. One brother had died of peritonitis, and a sister of exophthalmic goitre. The patient had always been in comfortable circumstances. She had enjoyed good health throughout most of her life, but had once been in the Royal Infirmary for a few weeks, on account of hæmoptysis. The pains for which she sought advice had troubled her for three weeks. On making a routine examination of the physical condition of the patient, it was found that with the exception of the circulatory organs every system presented phenomena in all respects normal. No symptoms were present that could be referred to the heart.

On inspection, a slight oscillation was seen in the veins of the neck, preceding in time the pulsation of the carotid arteries. The patient was

somewhat plump, and no impulse of any kind could be seen in the præcordia. Palpation determined that the apex beat was in the fifth intercostal space, 3 in. from mid-sternum. It revealed in addition another fact, viz., that there was a very distinct thrill preceding the cardiac impulse, and not confined to the region of the apex, but felt widely spread in every direction round that area. The radial pulse, 76 per minute, was full, of moderate tension and perfect regularity. The upper limit of

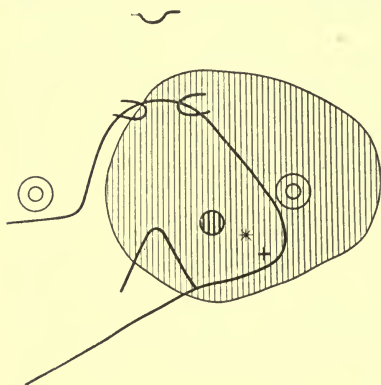


FIG. 169.—Tricuspid and mitral presystolic murmur in Case 44.

cardiac dulness was found to be at the level of the upper border of the third costal cartilage. The right border was  $2\frac{1}{2}$  and the left  $3\frac{1}{2}$  in. from the middle line. On auscultation, a loud rough presystolic murmur was heard over an area larger than, and embracing the whole of, the præcordia. The point of maximum intensity was exactly at the junction of the fourth left costal cartilage with the sternum, and from this spot it was conducted in every direction to different distances. It was audible  $3\frac{1}{2}$  in. upwards,  $3\frac{1}{2}$  in. downwards,  $4\frac{1}{2}$  in. horizontally to the right,  $5\frac{1}{2}$  in. horizontally to the left,  $4\frac{1}{2}$  in. diagonally downwards to the right, and 6 in. downwards to

the left. The facts are shown in Fig. 169. Although the maximum intensity was at the junction of the fourth left costal cartilage with the sternum, there was very little change in loudness for about a couple of inches in every direction around this spot; the murmur, for instance, was almost equally loud at the ensiform process and near the cardiac apex. There was absolutely no change in the character of the murmur when the mitral and tricuspid areas were carefully compared. The first sound was everywhere perfectly closed, the second sound was reduplicated, and the pulmonary element was both later in time and relatively louder than the aortic. After a few days' rest and treatment by means of salicylate of sodium, the patient was freed from her symptoms.

Now the results of auscultation in this case—which are shown in the tracing—are not absolutely free from the possibility of different, even antagonistic, interpretation. It may be held, on the one hand, that the presystolic murmur was entirely due to obstruction of the tricuspid orifice, or on the other, that it was caused solely by obstruction of the mitral orifice. It seems to me that the possibility of such a view as this latter hypothetical opinion may be at once dis-



missed, for if, with a presystolic murmur having its point of maximum intensity in the situation above described, there should be no tricuspid stenosis, "we shall," as Gairdner has so well put it, "have to rewrite our whole cardiac diagnosis and pathology of murmurs; for it is impossible to find a stronger case than this for the *absolute* diagnosis of tricuspid obstruction." This reasoning was strongly urged by me when the case was previously published.

No doubt another consideration supported the view that in this case there was a double lesion. If diagnosed as at any rate in part one of tricuspid obstruction, the rarity of this lesion without coincident mitral obstruction is a strong argument, when unopposed by the facts of physical diagnosis, in favour of combined mitral and tricuspid obstruction.

With reference to this very interesting case, one final remark must be made. It will be remembered that, with the sole exception of an attack of hæmoptysis on a former occasion when she was an inmate of the Royal Infirmary, her health has always been good. She has, in particular, never had a rheumatic symptom unless the pains for which she presented herself in November are of this nature, and it seems possible that the valvular lesions might have been congenital.

From the total absence of any symptoms, the obstruction was probably caused by some roughening of the auricular surfaces of the auriculo-ventricular valves, without any considerable narrowing of the orifice, and there could not be any interference with the closure of the valves, as there was no evidence of regurgitation.

Some time afterwards the subsequent events of the patient's life came to my knowledge. She was seized on 1st February 1893 with acute pleuro-pneumonia affecting the base of the left lung, and was attended by Dr. Robert Thin, to whom I am indebted for the information. The attack was very serious from its beginning, and the pneumonic consolidation rapidly extended so as to invade almost the whole of the lung. On the morning of the 6th, cedema of the other lung set in rapidly, and she died the same evening. Unfortunately no post-mortem examination was obtained.

The two cases (Nos. 43 and 44) which have been narrated

offer in certain respects a marked contrast. In the first, there was not only incompetence of both auriculo-ventricular valves, shown by the distinct murmurs of systolic rhythm in the tricuspid as well as mitral area, but an easy means of differentiation of the mitral and tricuspid presystolic murmur was afforded by their separate points of maximum intensity. This case also differed from the second in the presence of some definite results of valvular disease, *i.e.* the venous congestion of the lungs, and the œdema of the lower extremities. The second case, which has been previously discussed by me, presented considerable difficulty on account of the maximum intensity of what almost seemed to be a single presystolic murmur, and this murmur was absolutely free from anything tending to point in the direction of regurgitation.

### TRICUSPID INCOMPETENCE.

Regurgitation at the tricuspid orifice, formerly regarded as infrequent, is now admitted to be the most common valvular affection. According to Stewart Stockman, it is very common in the lower animals. There is a natural provision whereby some regurgitation is allowed at the tricuspid orifice when the intracardiac pressure on the right side of the heart is excessive. It appears to have been first observed by Hunter, and, as was mentioned previously, he based his opinions upon the results of injections. Little notice was taken of these views until Adams investigated the subject and extended the observations. To him is due the merit of showing clearly that what is necessary towards the maintenance of the systemic, would be injurious to the pulmonic circuit, where so many causes would temporarily retard the passage of blood through the lungs. Adams appears to have been the first to point out that the natural tendency to incompetence of the tricuspid valve saves it from injuries, to which the mitral valve is, from its unyielding nature, exposed. Wilkinson King published some most luminous observations upon this subject, and explained the mechanism, or "safety-valve action," by means of which he considered the regurgitation to be allowed. These views were at one time stoutly opposed, especially, perhaps, by Blakiston

and Walshe, but this natural function of the tricuspid valve is now almost universally admitted.

Tricuspid incompetence naturally falls into two great classes. The first group includes instances which, on account of some lesion connected with the valves themselves, may result from endocarditis or degeneration. The second group includes cases in which there is incompetence as the result of some affection of the muscular substance of the heart. The first-mentioned group is numerically small as compared with the second, to which belongs the overwhelming proportion of instances of tricuspid regurgitation. It therefore follows that the two groups stand to each other in well-marked antithesis.

ETIOLOGY.—Tricuspid insufficiency having its origin in purely valvular lesions may be produced by acute, subacute, or chronic endocarditis. It may, on the other hand, be produced by degenerative processes. In this class, the lesions of the tricuspid valve are very rarely isolated. Not merely is incompetence of this type associated, as a rule, with obstruction, but the tricuspid affection is linked with some other valvular lesion, more particularly with structural alteration of the mitral cusps.

The factors which lead to incompetence of the tricuspid valve in consequence of muscular changes are extremely numerous. It is worthy of note that this form of incompetence may be the sole cardiac affection present, and yet it can never, in any sense, be regarded as primary; it is on the contrary invariably consecutive to some other condition.

Tricuspid incompetence resulting from dilatation of the right ventricle may be brought about by causes acting mechanically upon the muscular walls. It is obvious that the lesions acting most directly upon the right ventricle, which may, in consequence, produce dilatation with incompetence of the tricuspid valve, are connected with the pulmonary artery. Obstruction of the pulmonary orifice or incompetence of its valve always leads to changes in the muscular substance of the right ventricle, producing hypertrophy, with or without dilatation according to circumstances. As pulmonary lesions are of but rare occurrence, they cannot be regarded as common causes of tricuspid incompetence.

With the exception of lesions of the pulmonary orifice, the affections which act most immediately upon the right ventricle are connected with the lungs. It is worthy of note that the disorders which lead in this way to most disturbance of the functions of the right ventricle, are those interfering with the distribution of the pulmonary artery, or the return of the blood by the pulmonary veins. It is, therefore, emphysematous and fibroid changes which lead directly to disorders of the right ventricle.

Bronchial affections, unless they cause interference with the access of air, have but little influence directly upon the right ventricle. Their blood supply is derived from the bronchial arteries which are of aortic origin; but inasmuch as chronic diseases of the bronchial tubes usually lead not merely to dilatation of the tubes, but also to emphysematous changes in the lung tissue, they in a more remote manner also cause dilatation of the right ventricle and tricuspid insufficiency. The only form of pulmonary phthisis which leads to such changes in the right ventricle is chronic fibroid phthisis.

Mitral lesions, by interfering with the circulation in the lungs, produce the same effects. It is a difficult matter to assess the relative influence exerted by obstruction and incompetence at the mitral orifice in the production of tricuspid incompetence; but this is a matter of less moment, seeing that the two affections are so frequently conjoined. Aortic diseases, in the absence of any mitral complication, have absolutely no influence over the right ventricle, for, so long as the mitral cusps are competent and the cardiac hypertrophy adequate, there can be no backward pressure upon the pulmonary circulation. When, however, in consequence of the heart outgrowing the nutritive possibility of the coronary arteries, the left ventricle becomes enfeebled, there is, as a consequence, some backward pressure upon the lungs, and, as a result, interference with the right ventricle.

When there is, as the result of hindrance to the passage of blood through the kidneys, increased pressure upon the left side of the heart, the left ventricle frequently becomes dilated and permits venous stasis in the lungs, and, therefore, interference with the right ventricle. It must not be for-



gotten, however, that here the pathogeny is somewhat more complex. There can be no doubt that degeneration of the muscular substance of the heart in cases of renal disease may be brought about by faulty nutrition, consequent upon the deterioration of the blood, and there is, further, a possibility of degeneration of the coronary vessels which will still more interfere with the nutrition of the heart.

In addition to such mechanical causes of interference with the right ventricle, leading to tricuspid incompetence, there are a great many factors of a more general nature which lead to analogous results. The causes which are operative in this way will be looked at in greater detail in the consideration of diseases of the myocardium; but to complete the subject of the etiology of tricuspid incompetence, it is advisable in this place briefly to refer to a number of causes falling under this group.

Pyrexia, if of more than brief duration, almost invariably leads to dilatation of the right ventricle and tricuspid incompetence. It does so sometimes from simple relaxation of the muscular substance, but in other cases by means of hyaline degeneration. Toxic influences belonging to almost every class produce the same effects; the toxins produced by micro-organisms (sometimes in the absence of all pyrexia), the organic poisons, such as alcohol, the inorganic poisons, such as lead, act in precisely analogous fashion. Malnutrition, whether arising from some morbid process, as malignant invasion, from deficient absorption, as in such a simple affection as dilatation of the stomach, or from some deficiency of food,—all lead to the same end. A long experience of out-patient service in our great hospitals enables me to bear witness to the extreme frequency of tricuspid regurgitation in atonic conditions of the stomach. Such disorders as anæmia, in which the nutritive power of the blood is lowered, are also to be considered as potent causes of tricuspid incompetence.

MORBID ANATOMY.—In those cases in which tricuspid incompetence is of valvular origin, there are anatomical alterations of the cusps analogous to those of the mitral valve. The normal thinness and translucency of the cusps may be replaced by thickening and induration. Such are the

most usual appearances shown by the cusps when they have suffered from endocarditis, and thickening and roughening of the chordæ tendineæ frequently attend upon these changes. In addition to such alterations, however, there are sometimes vegetations. These are more frequent in the endocarditis of early life.

Degenerative changes are sometimes to be seen presenting characters similar to those already described in connection with the mitral cusps. These degenerative changes, whether fibrous or calcareous, produce considerable distortion of the cusps resulting in their incompetence.

In cases of tricuspid incompetence it is much more common to find no structural alteration in the cusps, the sole alteration consisting in more or less dilatation of the auriculo-ventricular orifice. Further than this, there may not be any perceptible widening of the orifice, and the cause of the regurgitation must be assumed to lie in dilatation of the ventricle, causing want of adaptation of the valvular segments.

The associated alterations found in tricuspid incompetence are for the most part confined to the right auricle, which is sometimes dilated to an enormous extent. Sometimes there is hypertrophy of its walls in whole or in part. Instead of hypertrophy it is not at all uncommon, however, to find the thickness of the auricular walls considerably reduced. The great veins which empty their contents into the right auricle are always more or less dilated. The superior vena cava and its tributaries are sometimes so enlarged as to resemble those of aquatic mammals, and the valves which guard the entrance to the veins of the neck are incompetent. The inferior vena cava is even more dilated, but at the point where it passes through the diaphragm it is necessarily allowed less scope for distension.

The more distant effects are seen in general venous stasis with subcutaneous cedema, serous transudations, mucous catarrhs, and hypostatic congestion. The liver in particular suffers in tricuspid incompetence, and is often found to be greatly enlarged.

**SYMPTOMS.**—In slight cases of incompetence there may be a total lack of all subjective symptoms, but when there is any considerable degree of regurgitation the patient becomes

conscious of some of its effects. The symptoms which may be noticed are swelling of the ankles at night, feelings of heaviness in the right hypochondrium, some digestive troubles, such as dyspepsia, and diarrhœa along with scanty urine.

Headache is a common symptom, and only less frequent is a sensation of giddiness. Patients who suffer from high degrees of tricuspid incompetence are also prone to suffer from various disturbances of sleep. It is apt to be broken by frightful dreams, from which the patient awakes with a start, or insomnia may make its appearance, with the peculiarity that while the patient is unable to obtain repose at night a balance is attempted by means of a soporose tendency during the day. Further alterations in the functions of the brain are sometimes in evidence — illusions, hallucinations, and delusions being by no means uncommon.

A tendency to cyanosis is common in all cases of tricuspid incompetence, and in serious cases it forms one of the most marked features. Sometimes in consequence of venous stasis in the liver, and disturbance of the functions of this organ, the cyanosis is associated with a certain amount of jaundice, and when this is the case a somewhat singular greenish tint is the consequence. Dyspnoea is almost always present on exertion, and in grave cases it is a distressing feature. It must be ranked as connected with the causes as well as the effects of tricuspid regurgitation. The fact must never be overlooked that right-sided disturbances are more likely to produce interference with the functions of the pleura than affections confined to the left side of the heart, inasmuch as the blood circulating in the pleural membrane is in overwhelming proportion returned to the heart by the bronchial veins, which discharge their contents on the right side by means of the vena azygos, and on the left side by means of the superior intercostal veins. Their destination is therefore the right auricle. When disturbance of the functions of the right side of the heart occurs, there is as a consequence considerable liability to backward pressure upon the pleural membrane resulting in hydrothorax, the various factors of which have previously been fully discussed. This is the cause, in a considerable proportion of instances, of persistent

breathlessness which is often found in cases of tricuspid regurgitation. In the same way, and by the same mechanism, long-continued tricuspid escape tells upon the bronchial veins, leading to catarrh as the result of hyperæmia.

Swelling of the ankles is extremely common, but, except in serious cases, it disappears when the patient retains the recumbent posture. In grave cases, however, it is persistent, and is apt to be associated with anasarca of all the dependent subcutaneous textures. In severe cases an enlargement of the abdomen may be produced by the ascites resulting from backward pressure, and it need hardly be added that physical examination occasionally reveals the presence of fluid in the abdomen in the absence of all apparent enlargement. The area of hepatic dulness is enlarged in most of those cases which give rise to venous stasis, and there is not merely such an enlargement as may be made out by percussion, but an enlargement which can be detected by palpation. The enlarged liver is not infrequently tender to touch, as well as subjectively painful. The quantity of urine, as has already been remarked, is diminished, and on examination it is found to be deeper in colour and of higher specific gravity than in health. The amount of urea, although diminished as a rule, may yet be relatively increased as regards the amount per ounce of urine. Albumin is very frequently present, sometimes accompanied by blood, but more frequently by tube casts of different kinds, the most common amongst which are hyaline and blood casts. It occasionally happens that hæmatemesis and melæna occur as the result of backward pressure upon the portal system.

The radial pulse in tricuspid incompetence gives in itself little evidence of any circulatory disturbance. It is usually of smaller volume and of lower pressure than is the case in health. In the purely functional cases of tricuspid regurgitation the rate and rhythm of the pulse undergo no necessary alteration unless the tricuspid incompetence is the result of some disturbances of the left side of the heart, to which are to be attributed the alterations in the character of the pulse.

Examination of the neck frequently furnishes most characteristic appearances connected with the cervical veins. It



has already been shown that the jugular veins may present a considerable number of interesting phenomena whenever the continuous flow of blood from the veins to the heart is interrupted at its entrance into the right cavities of the heart. As this subject has been fully discussed in a previous section, it is unnecessary to do more than refer to the appearances which present themselves. In tricuspid incompetence any of the various conditions of the veins which have been described may be present, from a slight fulness showing respiratory influences and circulatory movements, to great turgescence, in which the veins stand out in a condition of profound distension as if they would burst.

On examination of the body abnormal impulses may be observed. The most common is an undulation of the epigastrium, produced, so far as can be seen, by the movements of the enlarged right ventricle. Much less commonly, impulses are to be seen in the third and fourth right intercostal spaces close to the sternum. These are occasionally caused by the contraction of the right auricle, but are more commonly the result of ventricular systole, and are due to the reflux of blood from the ventricle into the auricle. Palpation reveals no additional facts, and simply supports the evidence derived from inspection. The area of cardiac dulness is more or less enlarged bilaterally, but more particularly towards the right. Auscultation furnishes in a very large proportion of cases of tricuspid incompetence, more particularly of the functional variety, the sole evidence of the lesion. Yet it must be remembered that in many cases of tricuspid incompetence there may be no murmur, although other facts prove that incompetence is present. When the safety-valve action has undoubtedly developed into pronounced incompetence, as proved by the appearances in the veins of the neck, there may be a total absence of any evidence on auscultation. Even in such cases, however, it is very common to find that the first sound in the tricuspid area is somewhat weak, and it is not uncommon to find an apparent doubling of the first sound. The characteristic murmur heard in most cases of tricuspid incompetence is systolic in rhythm, while its character is soft and blowing. Its point of maximum intensity is usually about

the junction of the fifth and sixth left costal cartilages with the sternum, but this is subject to slight individual differences. It is propagated from its point of maximum intensity in every direction, and it sometimes requires considerable care to distinguish it from aortic and pulmonic murmurs.

The condition of the second sound in the pulmonary region is subject to considerable variations. It might naturally be expected that in those cases in which tricuspid incompetence takes its origin from some disease of the left side of the heart, or from some interference with the passage of blood through the lungs, the resulting accentuation of the pulmonary second sound would be lessened when tricuspid incompetence occurs. That this does take place can be easily determined by any one who watches the progress of such a case. The question, however, is a matter of degree, and in such cases, even after regurgitation has been established at the tricuspid orifice, there may nevertheless be accentuation of the pulmonary second sound, and also be a doubling of this sound at the base of the heart. So long as the pressure in the pulmonary circuit remains relatively increased, while that in the systemic circulation is relatively lessened, there must be a tendency to such accentuation or doubling. This has been very distinctly shown by Balfour.

DIAGNOSIS.—In slight cases of tricuspid incompetence there may be no evidence of the lesion beyond that furnished by auscultation, and it is precisely in such cases that there may be some difficulty in determining its nature. A simple systolic murmur heard in the tricuspid area may be produced at the right auriculo-ventricular orifice, or it may be propagated to that area from one of the other regions. If care be taken, however, in regard to the position of maximum intensity there should be no difficulty in recognising the exact significance of such murmurs.

In cases accompanied by venous phenomena in the neck there is never any difficulty in arriving at a correct conclusion, as the association of such symptoms with enlargement of the right side of the heart, added to the evidence furnished by auscultation, will afford diagnostic indications which cannot be mistaken.

Even in the entire absence of any murmur in the tricuspid area regurgitation may be determined by free venous pulsations in the neck, as has been held by almost all the important writers on cardiac affections. Complete references to these authors will be found in a contribution to the subject by myself.

PROGNOSIS.—The prognosis in cases of tricuspid incompetence must be based upon the general conditions which are present. In those instances which will be afterwards dealt with in connection with disorders of the myocardium, where the tricuspid incompetence is produced by some temporary weakness of the wall of the ventricle, the prognosis is excellent. The same may be said of cases in which regurgitation occurs as the consequence of some temporary interference with the functions of the lungs. It is far otherwise, however, as regards tricuspid regurgitation as the sequel of disease of the left side of the heart, since when the right side of the heart has failed, the reserves may be said to have given way.

TREATMENT.—But little requires to be said in regard to the special treatment of tricuspid incompetence. The main lines of treatment suggested for valvular lesions in general must be followed, but there will require to be more especial care as regards the effects on the systemic veins. In those cases in which the affection is the result of mitral lesions, treatment must in the first instance be devoted to such symptoms as may be present. When the tricuspid incompetence is produced by some pulmonary disorder appropriate measures for relieving the pulmonary troubles must be adopted. If the incompetence is produced by relaxation of the ventricle from some general cause, such as pyrexia or anæmia, the treatment should be directed towards the relief of the primary affection. When, as is very often the case, tricuspid regurgitation has its starting-point in faulty digestion, more particularly chronic gastric catarrh and atonic dilatation of the stomach, these factors require to be eliminated. If, lastly, the primary cause be cardiac strain from overstress, the influences which have produced the condition must be removed. In all cases the use of cardiac tonics must be adopted. It is often said that digitalis and its allies have but little effect on the right ventricle. Morison, for instance, speaks of them

as not only useless but dangerous. These drugs, nevertheless, have proved, in my experience, in all remediable instances of tricuspid incompetence of the greatest service, and cannot be too strongly recommended.

In those instances presenting symptoms of enlargement of the liver, and gastro-enteric catarrh, the combination of digitalis and mercury will be found most useful. To these is sometimes added squill, but this is of more doubtful utility. When there is gastric dilatation, nux vomica along with digitalis or strophanthus can be confidently recommended. In all such cases, when there is any digestive inadequacy, the regulation of the diet must be a prime consideration.

The treatment of tricuspid incompetence from cardiac strain and analogous conditions will be referred to in the chapter on myocardial conditions.

CASE 45. *Cardiac Dilatation and Tricuspid Incompetence from Malaria.*—J. B., aged 24, cab driver, married, presented himself as an out-patient at the Royal Infirmary, 4th March 1891, complaining of pain in the region of the abdomen. There had been no hereditary tendencies to disease. The patient's father and mother were perfectly well, as were also all their children, with the exception of one daughter who had died of cerebral abscess, in consequence of ear disease. The social conditions had always been satisfactory. His health had been good until he suffered from a severe attack of malaria while in Massachusetts during the year before he was seen. The pain for which he sought advice began four months before he did so, and had continued ever since. He was somewhat anæmic in appearance, with pallor of the lips, gums, and tongue, as well as of the conjunctivæ. The alimentary system was obviously free from any disturbance. The organs were all of their usual size. There was no enlargement of the spleen. The hæmoglobin was 50 per cent.; the red blood corpuscles numbered 4,230,000; there was no increase in the number of the white corpuscles. The blood contained a considerable number of pigment granules, and there was some alteration in the appearance of the red blood corpuscles, there being large and small varieties, as well as many with a change in shape. No organisms were detected.

The radial artery was healthy, the vessel moderately full, and the pressure low. The pulsation was perfectly regular, its rate was 86, each wave was large, bounding, and dicrotic. There was well-marked jugular pulsation in the neck. The apex beat was in the fifth intercostal space, 3 in. from mid-sternum. There was a visible pulsation in the third and fourth left intercostal spaces. No thrill could be elicited on the application of the hand. The area of cardiac dulness extended  $2\frac{1}{2}$  in. to the right



and  $3\frac{3}{4}$  in. to the left of mid-sternum at the level of the fourth costal cartilage.

On auscultation there was a loud venous hum in the neck, and in the standing or sitting posture a very faint systolic murmur was audible at the junction of the sixth left costal cartilage with the sternum. No other abnormal symptom could be detected in these positions, but when the patient lay down the tricuspid murmur became loud, harsh, and almost musical. It still retained the same position of maximum intensity in the tricuspid area, but in the recumbent posture it was distinctly propagated up the sternum almost as far as the aortic and pulmonary areas, but it was not conducted outwards on either side of the sternum.

There was no symptom of disease connected with any other region, excepting that the patient was decidedly weak and lacked energy.

Under treatment with arsenic and digitalis the patient rapidly improved, the first evidence of this being an increase in the intensity of the murmur and an enlargement of its area of audibility. This, however, after a few weeks gave place to gradual disappearance of the murmur altogether, and the blood at the same time showed a return to health.

There could be no doubt that in this case the toxic effects of the malaria by acting on the blood had interfered with its nutritive possibilities, and in this way brought about the cardiac dilatation.

CASE 46. *Tricuspid Incompetence from Bronchial Affection.*—A. N., aged 44, widow, engaged in household duties, was admitted to Ward 25, 4th April 1892, suffering from breathlessness.

Her family history showed no hereditary disease tendencies, and her social condition had always been fairly satisfactory. Her health had been good until the last two or three years, during which she had suffered a good deal from winter cough, accompanied by breathlessness. She had a furred tongue, little appetite, and constipation on admission. The jugular veins were seen to be turgid on inspection, with slight oscillations in them, occurring both with the respiratory movements and with the auricular systole.

The apex beat was invisible, but it could be felt on palpation in the fifth intercostal space, 4 in. from mid-sternum. Palpation revealed no thrill or other abnormal phenomena. On percussion the right border of the heart was found to be 3 in., and the left  $4\frac{1}{2}$  in. from mid-sternum. On auscultation in the mitral area there was a distinct doubling of the first sound; in the tricuspid area this double first sound was also present, but it was immediately followed by a soft-blowing systolic murmur, having its maximum intensity at the union of the sixth left costal cartilage with the left edge of the sternum, and propagated upwards as far as the third costal cartilage. In the aortic and pulmonary areas the first sound was unaccompanied by any murmur. The second sound was distinctly doubled, and the later of the two sounds—much louder than the one which preceded it—had its maximum intensity in the pulmonary area.

The patient's respirations were hurried, varying from 35 to 40 per minute at the time of her admission. All the respiratory muscles were brought into play. There was a great deal of rhonchial thrill, but no increase of vocal fremitus. On percussion the chest was perfectly clear throughout, except towards the bases of the lungs behind where the sound was slightly muffled. The breath sounds were absolutely obscured by sonorous and sibilant rhonchi of every pitch, while at the bases there were many crepitations. The vocal resonance was unaltered.

The use of carbonate of ammonia, spirit of chloroform, and infusion of digitalis, speedily brought about great improvement. The cough and dyspnœa speedily disappeared. The physical signs of the bronchial and pulmonary affections passed away, while the doubling of the cardiac sounds and the systolic murmur gradually became less perceptible, until, when the patient was discharged, no abnormal sound was present save a faint systolic tricuspid murmur. The area of cardiac dullness was also reduced to such an extent that the right border of the heart only reached 2 in. and the left 4 in. from mid-sternum.

This was an excellent example of implication of the right side of the heart and of its great venous orifice in consequence of bronchial catarrh, the mechanism not being direct but acting through increasing the intrathoracic pressure by forced expiratory efforts.

CASE 47. *Tricuspid Incompetence from Mitral Disease.*—M. D., aged 23, domestic servant, was admitted to Ward 27, 18th October 1897, complaining of pains in the chest and want of breath. Her father had died of heart disease following acute rheumatism; her mother of cancer. She had two brothers, both alive and well; there had been one sister, who died in infancy. The patient had suffered from some of the febrile infectious diseases of youth, and she had also been for some time ill with chorea when 10 years old. The illness for which she came under treatment began about five years before admission, in an insidious manner.

The patient complained of pain radiating from the left præcordia to the left shoulder and arm, often to the finger-tips, and of much breathlessness. The alimentary functions were satisfactory. The liver was, however, considerably enlarged, extending from the fourth rib downwards for 6 in. in the mammary line, and blending with the splenic dullness in the left hypochondrium. There was no ascites. The glands were in no way affected. The hæmoglobin was 70 per cent., and the red blood corpuscles numbered 4,000,000.

A slight degree of cyanosis was obvious in the tint of the lips, but nowhere else. There was well-marked pulsation in the jugular veins, external and internal; a vein on the front of the right infra-clavicular area was also much distended and showed free movements. The pulsation in all these veins was of auricular rhythm. The apex beat was found in the sixth intercostal space 4 in. to the left of the mid-sternum. Pulsation

was also visible in the third, fourth, and fifth left intercostal spaces, but, what was more striking, there was also pulsation in the third and fourth right intercostal spaces. This latter preceded the systolic impulse and the movement to the left of the sternum. Palpation confirmed these facts, and also showed that the pulsation in the vein on the front of the chest was palpable. It further revealed a thrill at the apex, which began with the diastolic rebound and continued until the systole could be felt. The cardiac dulness extended  $2\frac{1}{4}$  in. to the right and  $4\frac{1}{2}$  in. to the left of mid-sternum. On auscultation in the aortic region the sounds were faint and a soft systolic murmur was audible, propagated from below. In the pulmonary area this systolic murmur was heard with much accentuation of the second sound. At the apex the cycle seemed to begin with a rough but not loud murmur running up to a soft-blowing systolic murmur which accompanied the cardiac impulse; neither first nor second sounds could be heard. By timing the rough murmur through the application of the hand to the præcordia it was found to begin with the diastole. In the tricuspid area there was a loud systolic murmur, which, although different from, yet blended with, the mitral murmur of the same rhythm. The radial artery was soft and yielding, being empty and showing low pressure; the pulse was about 80, fairly regular and equal. There was some comparative dulness of the left pulmonary base with crepitations. No disturbances of the urinary functions were present. The catamenia were regular but scanty. There was no œdema of the ankles, and the only nervous symptom was the pain in the chest. By means of absolute rest and cardiac tonics the patient speedily recovered, and left hospital in a few weeks. She has since that time, however, been again an inmate of my ward with the same symptoms.

This case is of interest in several directions, but, in view of the present connection, chiefly by reason of the pulsation of the vein on the front of the chest and the movements of the right auricle. The pressure of typical anginous symptoms is also worthy of note.

### TRICUSPID OBSTRUCTION AND REGURGITATION.

It is quite unnecessary to dwell upon combined lesions found at the tricuspid orifice. In their morbid anatomy they incline towards the appearances presented by obstruction; in their clinical features they may approach the results either of obstruction or of incompetence, but as the obstruction is often latent, the diagnosis of incompetence can alone be made. The cases recorded, placed for convenience under obstruction, have exemplified these remarks.

## CHAPTER XIV.

### AFFECTIONS OF THE MYOCARDIUM.

AMONGST ancient physicians it appears to have been the belief that the muscular substance of the heart was almost impervious to disease, and these great observers, Senac and Morgagni, must be regarded as having inaugurated a new era of thought regarding it.

Corvisart made many observations upon this subject, and followed Senac in regarding myocarditis as being specially linked with affections of the cardiac membrane. Latham and Craigie called attention particularly to the development of purulent myocarditis. Stein made great advances in regard to the intimate structure of the changes taking place in the disease, and Virchow threw much light upon some forms of pathological modification.

Many other authors whose work has been of real importance in connection with this subject may be mentioned—among them Greenfield, Goodhart, Weigert, Huber, and Lindsay Steven. Other valuable observations will be referred to in the sequel.

In considering the diseases of the myocardium, there are great difficulties in attempting to arrange them in anything like a scientific order. The most natural arrangement is undoubtedly to take up the various disturbances in the order of their intricacy; to begin with those which are more simple, to consider at a later stage those which are more complex, and finally, to discuss the changes which are in their origin obviously conservative or compensatory, but which are,



nevertheless, very frequently associated with important lesions. According to this plan the myocardial affections fall into the following groups:—simple cardiac debility, atrophic processes, degenerations, myocarditis, and neoplasms. These affections may lead to cardiac dilatation, aneurysm of the heart, and rupture of its walls, the consideration of which naturally follows. The entire subject naturally terminates with the conservative or compensatory changes, summed up under the term hypertrophy, to which must, lastly, be added a few remarks upon gross lesions resulting from injury.

A most important preliminary subject connected with the myocardium is the effect of interference with the coronary arteries. The distribution of these vessels has already been considered from the anatomical point of view; but there are some aspects of the subject which require attention from the standpoint of pathology. As has been well put by Letulle, there is something paradoxical at first sight in the facts of this distribution, inasmuch as the calibre of the left, or anterior, coronary artery, which is evidently intended to supply the left ventricle, is less than that of the right, or posterior, which passes round the base of the right ventricle. The apparent anomaly, however, is easily explained, seeing that the left coronary only supplies the anterior surface, the apex, and the left edge of the left ventricle, along with part of the inter-ventricular septum and a small portion of the anterior surface of the right ventricle. All the rest of the heart, that is to say, almost the whole of the right ventricle, the posterior aspect of the left ventricle, and nearly the whole of the auricles, are supplied by the right coronary artery.

The condition of the coronary circulation dominates, of necessity, the nutrition of the heart, and any interference with the lumen of the arteries, by lessening the supply of blood, leads to consequences immediate or remote according to the extent of the disturbance. The sudden stoppage of the circulation in any of the larger branches may lead to instantaneous death, as has been frequently recorded. The closure of one of the smaller branches may produce hæmorrhagic infarct if it occurs suddenly, or white softening if it is a more gradual process; and these conditions may lead to the formation of

cardiac aneurysm, or bring about a rupture of the walls of the heart. Gradual sclerotic processes produce ischæmia of the heart, and by interfering with nutrition lead to chronic fibroid changes in the myocardium which are probably in the first instance of a compensatory character. Sometimes fatty changes are the result, or fibroid and fatty conditions may be associated together.

Disturbances in the blood supply to the heart may occur independently of any change in the coronary vessels. In all conditions involving alterations in the relative blood pressures within the arterial and venous systems, the heart must necessarily suffer. If there be diminished arterial pressure, there will be a tendency to ischæmia of the heart-muscle, leading in many instances to structural alterations; and if there be at the same time any retardation of the return of blood from the coronary sinuses, there must infallibly be some venous stasis, leading to a cyanotic or cedematous condition of the myocardium.

The arrangement of the terminal branches of the coronary arteries has been the subject of much discussion. Swan suggested that the terminations do not anastomose, and this view was accepted by Quain and Cohnheim. Legg and West hold that there is comparatively free communication. Steven has carefully investigated the subject anew by means of injections, and shows that any communication is only by means of the finest branches. His conclusions are that the arterial inosculation is so trifling and distant as to be quite unable to compensate for vascular obstructions, except in their immediate neighbourhood, and even then only after very considerable destruction of cardiac tissue has occurred.

Erichsen, acting on an idea of Marshall Hall's, that interruption of the coronary circulation would cause rapid death, was one of the earliest experimenters upon the coronary arteries. Death took place within ten minutes from the commencement of the symptoms resulting from ligation of these vessels. Panum injected tallow into the coronary arteries, and noticed that the left ventricle ceased to beat seventy-five minutes, and the right ventricle ninety minutes, from the time of the injection.

With regard to the influence of the coronary circulation, Cohnheim mentions cases where the sole cause of death was embolism of the coronary arteries, and Osler deems this to be a common cause of sudden death. Many cases of the kind have been narrated, and Rolleston has recently recorded a case of sudden death due to embolism of the left coronary artery about three-quarters of an inch from its origin, just before the septal branch came away. Instances of this kind throw a lurid light upon the overwhelming influence of the coronary circulation over the muscular walls of the heart.

With regard to the incidence of affections of the muscular substance of the heart, the statistics of the Edinburgh Royal Infirmary for the past five years, as collected by Gillespie, show that of 2368 cardiac cases 284 were instances of myocardial affections—222 in males, and 62 in females.

### CARDIAC WEAKNESS.

Under this head attention must be directed to a group of cardiac affections neither sharply defined in themselves nor distinctly separated from other myocardial changes of a graver character. It is impossible to deny that conditions arise in which the heart manifests loss of energy without undergoing any definite structural changes, and it seems advisable to include such states under the term "weak heart." The phrase has become almost classical, not only in our own country but throughout Europe and America. It may accordingly be retained in the sense above referred to. One of the most useful contributions to the subject is to be found in the important work of Hayden.

ETIOLOGY.—It cannot be doubted for a moment that there is such a condition as congenital inadequacy of the heart. Some individuals are not endowed from birth with kidneys, or livers, or brains sufficient to carry on the average functions of life; in the case of the heart, there are also many who are deficient from birth in a similar manner. The organ may be equal to the demands made upon it during a quiet uneventful existence, but, when called upon to meet the requirements of any period of storm and stress, it is found wanting.

At first the effects produced upon such a weak heart by greater demands than it can fulfil are shown merely by feebleness of all its functions, but, if the requirements are continued, symptoms arise proving that dilatation is present, probably with some underlying degenerative change. Stress thus leads to strain.

The heart may become weakened by a large variety of causes. Many of these are identical with the morbid agencies which when more severe or longer continued give rise to the different atrophic and degenerative processes to be described in this chapter. It is not therefore necessary to dwell upon them at this time. Suffice it to say that, in addition to pyrexia, which is one of the most powerful factors in weakening the heart, and toxic influences, more especially alcoholic excess, any lack of nutrition, whether arising from faulty digestive processes, imperfect blood formation, defective tissue changes, or drains upon the system, may lead to weakness of the myocardium. Over and above such causes, the effects of continued over-fatigue must be regarded as efficient causes of cardiac debility. It is probable that such excessive fatigue does not only act directly on the heart by mechanical effects, but that by exhaustion of the cerebral tissues it also indirectly lessens the trophic functions of the vagus.

MORBID ANATOMY.—In certain cases of cardiac weakness there are no definite lesions which can be detected by the most careful examination, and the morbid state consists in a mere relaxation of the tissues. In such instances the tissues are softer, as well as more easily torn and crushed, than in health; but there is no structural alteration to be seen with the naked eye, and on microscopic examination the tissue is apparently healthy.

In many other conditions, however, which clinically would not be distinguished from those just mentioned, there are more or less extensive changes in the myocardium. There is, for instance, the fibrillary atrophy, more particularly studied by French observers, in which there is an exaggeration of the longitudinal striation of the muscular element. It is believed to be produced by a change in the interfibrillary substance, so that the fibrils tend to be separated from each other.



There is also the fragmentary atrophy, or segmentation, produced by necrosis of the myosin. Granular atrophy is also found, with diminution of the striation without loss of substance, and it contains a large amount of granular material; this is often found accumulated in a longitudinal arrangement at each end of the nucleus. Mention must also be made of pigmentary atrophy, in which more or less pigment, doubtless having its origin in the hæmoglobin, is distributed in the muscle cell around the nucleus, somewhat in the same way as in the case of granular atrophy, so that it almost seems to prolong the extremities of the nucleus. These masses of pigment are of a brownish hue. A certain amount of pigment is normally present in every heart after youth has been passed, and it is by no means easy to say what quantity is to be regarded as abnormal. Letulle points out that all causes of nutritive disturbance usually give rise to some pigmentation, that, in short, a senile condition of heart, whether normal or abnormal in its relation to age, is shown by more or less destruction of the hæmoglobin of the muscles. Such changes are always most marked in that part of the heart which suffers the greatest amount of strain.

Degenerations of the cardiac muscle are frequently found to be the lesions revealed clinically by cardiac weakness. Nothing need be said on this subject in this place, as they are accorded a section to themselves.

**SYMPTOMS.**—The clinical features of cardiac weakness are often superadded to others, already present, characteristic of such general or local diseases as have given rise to the debility of the heart, and they are therefore apt to be overshadowed by them. The symptoms belonging to the cardiac condition are simply breathlessness and palpitation, such as may occur in any condition interfering with the energy of the heart, with occasional faintness and giddiness; but there may be subcutaneous œdema, and accumulation in the serous cavities. The pulse is usually empty, and the pulse wave small. The rate is subject to great differences, sometimes being very infrequent and at other times extremely frequent. The rhythm is apt to become irregular. The cardiac impulse is feeble or imperceptible. The area of cardiac dulness may

be absolutely normal, or it may be enlarged, in which case the feebleness of the muscle has led to dilatation. The heart sounds are weak, so much so as often to be almost imperceptible, and the first sound invariably suffers more than the second. It is extremely uncommon to find that all the cardiac sounds have disappeared, but in severe acute cases of disease the first sound is often entirely obliterated. There may, on the other hand, sometimes be soft blowing systolic murmurs entirely replacing the first sound in the mitral and tricuspid areas.

Along with such local appearances there may be others which result from them as consequences. Amongst these are venous stasis, with all its effects, whether hyperæmic or cedematous, and, in consequence of these, great interference with all the functions of the body.

This appears to be the most suitable connection in which to discuss the much-debated appearances found in many conditions of cardiac debility. The symptoms have been adverted to in the general analysis of circulatory phenomena, but, since they belong to weakness of the heart, this is the proper place for their fuller elucidation.

Several years ago considerable interest was shown in certain discussions as to the cause of the clinical facts observed in cases of cardiac debility. The principal subjects under debate at that time were the explanations which had been advanced regarding two of the phenomena commonly observed in feeble conditions of the heart. The first of these, and that the more frequent in its occurrence, is the systolic murmur heard in the second left intercostal space, at or near the pulmonary area; the second, not so often presenting itself to the observer, is the systolic impulse seen and felt in the same locality. To the investigation and explanation of these appearances several observers devoted much attention, and many of the points connected with the physical signs under discussion were virtually settled. As one who took part in the discussions on this question, it seems to me nothing more than simple justice to those whose views then differed from my own to state frankly and candidly the opinions which have been borne in upon me since the time referred to. In attempting to do so, it will be well to avoid unnecessary

reference to older observers. This may be done the more easily, as Russell has given a complete and masterly summary of the views of previous authors in his work on this subject.

For the present purpose it is only necessary to recall a few facts. In order to account for the systolic murmur and accompanying pulsation sometimes seen in the second left intercostal space, in cases of mitral incompetence, Naunyn advanced the hypothesis that both appearances are produced by the backward stream from the left ventricle into the left auricle. According to this view the systolic murmur is of mitral origin, and is conducted by the regurgitant current into the dilated left auricular appendix, while the pulsation is caused by the same stream distending the appendix and thereby producing an impulse on the thoracic parietes. Balfour applied this hypothesis to the corresponding phenomena so commonly seen in the feeble heart of anæmia and allied conditions. In his work on diseases of the heart, as well as in separate papers dealing with this special question, he has strongly advocated this explanation, and his opinions were warmly supported in some contributions made at the same time by myself. We were, however, unable to adduce any evidence obtained from morbid anatomy in favour of our views, and although many of the clinical features appeared to be explained by them, they could not be regarded as resting on any sure pathological basis.

Russell brought to the elucidation of the questions under discussion a large number of clinical and pathological observations, from the consideration of which he came to very different conclusions. He showed that, in many cases where the systolic pulsation in the second left intercostal space had existed before death, post-mortem examination proved that the impulse could only have been caused by the *conus arteriosus*, which, in consequence of dilatation of the right ventricle, was so far to the left as to occupy the site of pulsation in the left intercostal space between the second and third cartilages. With regard to the basic murmur, heard in cardiac debility, Russell proposed two explanations. He suggested that in some cases it might be produced by dilatation of the left auricle, which, pressing upwards upon the

pulmonary artery, gives rise to a narrowing of its lumen, while in other cases it is simply the systolic murmur of tricuspid incompetence propagated upwards to the conus arteriosus.

The main points at issue in the discussions regarding this subject were very critically examined and judicially weighed by Bramwell in his systematic work. As the result of a very careful review of the arguments which have been advanced by Balfour and Russell, he rejects the theories of both with regard to the production of the basic systolic murmur, and attributes it to the sudden pulsation of a large blood wave of abnormal composition into the vessel, which he thinks may probably be dilated.

Handford holds that the pulmonary systolic murmur, which he describes as disappearing in the erect position and reappearing when the patient is recumbent, is produced by the pressure of an enlarged, flabby, and dilated heart on the pulmonary artery.

Foxwell has, like Russell, found the pulmonary artery to be displaced considerably upwards. He regards the murmur in the pulmonary area as caused by a complicated change in the shape and position of the pulmonary artery, whereby its curve becomes increased, its axis and that of the right ventricle lie at a different angle from that existing under healthy conditions, and the vessel is flattened against the aorta. At the same time, however, he accepts Russell's view of a distended right auricle as the cause of the murmur in some cases.

In the last place, Sansom, after an examination of the views of Balfour and Russell, which leads him to dissent from both, advances the opinion that the basic murmur can be initiated at the overstrained portion of the right ventricle, the conus just below the pulmonary valves, by the production of a fibrillar tremor. He is, however, also inclined to believe that the cusps may themselves vibrate in the current.

It is easy for me now to consider the questions involved in a perfectly dispassionate and impartial spirit, inasmuch as Russell has, in my opinion, disproved the views of all observers previous to himself. He has demonstrated that the left



auricular appendix does not reach the anterior wall of the thorax, and that the pulsation in the second left intercostal space is produced by the conus arteriosus. The observations of Foxwell, Harris, and Mackenzie support him in this, and it seems to me that the explanation of Naunyn and Balfour has thus been refuted.

This decision leads of necessity to the further conclusion that the hypothesis of Naunyn and Balfour with regard to the origin of the systolic murmur heard in the pulmonary area falls to the ground, for since the left auricle never touches the parietes there is no medium for the conduction of a mitral murmur towards the base of the heart. But it must further be stated that in a large proportion of cases there is no evidence of any mitral incompetence, and that it is a mere begging of the question to assume it.

The view advanced by Bramwell may be regarded as in every respect a compromise, as Russell puts it, between the explanations of Hope and Beau, and it has, like their theories, been effectually disposed of by him.

But while granting freely that Russell has disproved all previous theories, it seems to me that part of his own explanation will not bear investigation. He has yet to prove that in early stages the left auricle is dilated. In truth, the conditions appear to be the very reverse of those which he postulates. The mitral cusps are often perfectly competent, and as long as there is no mitral regurgitation the pressure in the pulmonary artery must be greater than that in the left auricle.

The view of Handford cannot be accepted as a probable explanation for most cases, not only because the basic murmur is heard very frequently indeed while the patient is in the erect position, but also because it makes its appearance before there is any noteworthy enlargement of the ventricles. The same argument applies with equal cogency to the reasoning of Foxwell, while his experiment of forcing water into the right ventricle of a debilitated subject after tying the pulmonary artery is so unlike anything in nature that it cannot be held to prove anything.

Sansom, finally, is obviously in error, as, if overstrain of

the ventricle were a valid cause for a murmur, such a phenomenon would be of much more common occurrence than is the case. Almost every case of chronic renal cirrhosis, for example, would be attended by a murmur produced by the strain thrown on the left ventricle.

My own view is that the impulse in the second left intercostal space—the auricular impulse of Balfour—is produced by the conus arteriosus. The systolic murmur heard in the same position is, in my opinion, caused by tricuspid incompetence, but it is perfectly possible that for some cases Handford's explanation is admissible. The views to which my adhesion has been given were fully published within recent times.

DIAGNOSIS.—The recognition of the enfeebled condition of the cardiac muscle is easily reached. The characters furnished by the pulse, and the appearances ascertained on examining the præcordia, are sufficient to reveal the cardiac debility. This, however, is only one step in the determination of the nature of the affection. Any definite conclusion as to the exact structural change present can only be a matter of inference. The considerations which carry weight in attempting to form a probable conjecture as to the nature of the alteration are connected with the causes of the weakness and the general state of the patient. Conditions of malnutrition, bloodlessness, and pyrexia, when neither severe nor prolonged, are likely to produce simple relaxation of the muscular tissues, or some slighter degree of one of the atrophic processes; while profound cachexia, grave anæmia, and high fever, more especially if long continued, tend towards the degenerative changes, or chronic myocarditis. Youth is more liable to the less serious alterations—age to those which are more severe.

PROGNOSIS.—The prospects in simple cardiac weakness are usually good, so that, if the probabilities are in favour of a diagnosis of this condition, the prognosis may confidently be hopeful. The chief care must be directed towards the conclusion that the causes of the cardiac change and of the general state of the patient warrant the exclusion of atrophic and degenerative processes.

TREATMENT.—Simple weakness of the heart must be treated by meeting the causes of, and obviating the tendencies in, each

case. When arising in the course of any acute disease it passes away, with few exceptions, on the favourable termination of the primary affection. No special treatment is therefore necessary. As the result of alcoholism the condition also undergoes speedy improvement on removing the cause. If produced by symptomatic anæmia, or by chlorosis, cardiac weakness can be readily removed by the use of iron, and the general means of treatment employed in such cases. As it is too often but the precursor of serious structural lesions when occurring in the course of grave cachectic states, treatment cannot be of much avail; the usual methods in every class must nevertheless be employed.

In every instance the general lines of management for impaired cardiac energy must be adopted as regards rest, diet, air, and surroundings, while massage and cardiac tonics are of great advantage. It is in cases of this kind that baths and exercises produce their best results.

In order to present the clinical features under discussion in a concrete form, the following case is worthy of record. It has already been otherwise utilised in a previous chapter, p. 580.

CASE 48. *Cardiac Weakness from Pyrexia*.—Maggie G., æt. 18, unmarried, engaged in household duties, was admitted to Ward 25 of the Royal Infirmary on 5th June 1893, complaining of pains in her wrists and elbows.

Her father and mother, both æt. 42, had always been in good health. She had four brothers and one sister, all very strong, but three brothers had died in infancy. The patient's social surroundings had always been good. She had never been very robust, and four years before admission had suffered from a rheumatic attack, since when she had never felt very well. About four months before entering the hospital, pains had begun in the joints and had persisted ever since.

On her admission the patient was found to be somewhat pale, with a bright spot on each cheek. The skin was moist. The temperature was normal. The tongue was slightly furred, but the digestive system was otherwise healthy. There was no symptom connected with the hæmopoietic system. She complained of some palpitation and a slight degree of breathlessness. The pulse showed low pressure and moderate volume; it was perfectly regular, varying in rate from 80 to 90. There was some pulsation in the veins of the neck, and a very distinct impulse in the second left intercostal space. On palpation the apex beat was found to be in the fifth left intercostal space,  $3\frac{1}{4}$  in. from mid-sternum. The pulsa-

tion, systolic in time, in the second left intercostal space was found to be most distinct  $1\frac{1}{4}$  in. from the mid-sternal line. A tracing obtained from it by means of a revolving cylinder is given in the accompanying figure (Fig. 170). No thrill could be detected over any part of the præcordia. The cardiac dulness extended to 1 in. to the right and 4 in. to the left of the middle line at the level of the fourth rib. On auscultation, a venous hum was heard in the neck, and there were murmurs, systolic in rhythm, over the whole præcordia, which, on careful analysis, proved to be twofold. Around the region of the apex beat, and with its maximum loudness in the fourth interspace  $3\frac{1}{2}$  in. from mid-sternum, there was a harsh-blowing systolic murmur, conducted as far as the edge of the sternum to the right, and beyond the anterior axillary line to the left. Over almost the entire sternal region there was a soft-blowing systolic murmur, quite different in character from that heard at the apex. It had the same tone throughout

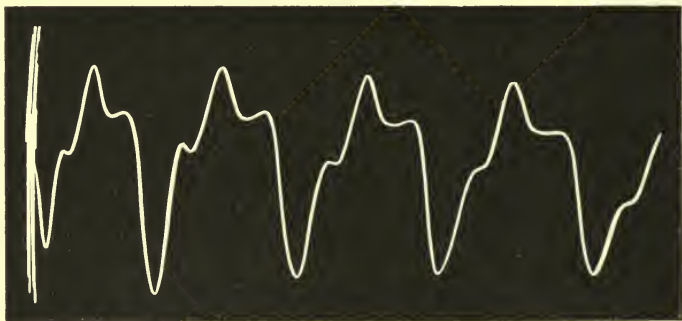


FIG. 170.—Tracing from Case 48.

the whole sternal region, but it seemed to have two points of maximum intensity—to be more exact, it was loudest in the pulmonary region, exactly over the area of pulsation, from which point it waned in its intensity in every direction until near the lower end of the sternum, when it became louder, again culminating at the point where the left side of the sternum was joined by the sixth costal cartilage; but in this situation the murmur was not quite so loud as over the area of pulsation in the pulmonary region. The second sound was frequently reduplicated, and the later of the two second sounds, which could be determined to be that due to the pulmonic cusps, was instantly followed by a short, sharp, high-pitched murmur, perfectly soft in character. This murmur was extremely restricted in its distribution, being only heard over a small triangular area  $2\frac{1}{4}$  in. in vertical and 2 in. in horizontal measurement, extending along the left border of the sternum, from the lower border of the third costal cartilage to the upper border of the fifth. This murmur was perfectly soft in character, and was absolutely unlike the obstructive diastolic murmur which is found in mitral stenosis. It could not be due to aortic disease, of which there was no indication, and



it could only, therefore, be a murmur of regurgitation from the pulmonary artery into the right ventricle, due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps. The production of this murmur has been discussed in Chapter XII. All these murmurs are shown in Fig. 171, in which the areas over which the murmurs were audible are distinguished in the usual way.

The other systems presented no symptoms of disease, with the sole exception of a few crepitations at the bases of both lungs.

The diagnosis was cardiac dilatation, with mitral and tricuspid regurgitation, produced by the febrile affection; it was nevertheless considered probable that some stenosis of the mitral orifice might be insidiously progressing, although this was a mere supposition, not based on any direct evidence. The crepitations at the bases of the lungs were regarded as the expression of passive congestion from mitral incompetence, and the diastolic murmur was assumed to be one of pulmonary escape, in consequence of the strain on the artery from the high pressure within it.

By means of salol and similar remedies the patient was relieved of her rheumatic symptoms, and the administration of iron with other tonics greatly improved the cardiac condition. The diastolic murmur disappeared, and the lungs cleared up, but at the time of the patient's departure from the Royal Infirmary, on 17th July 1893, she still had the systolic murmurs, and the pulsation in the second intercostal space. She presented herself at the hospital on the 2nd March 1894, when the diastolic murmur was found to be still absent, but the systolic murmurs were present as before. The first sound in the mitral area, preceding the systolic murmur, was, however, loud and clanging in character, which seemed to support the view that a stenosis of the mitral orifice was gradually developing.

This case brings into prominence the systolic impulse in the second left intercostal space, as well as the systolic murmur in the same position. From the physical signs there could be no doubt of the presence of mitral and tricuspid incompetence, and it may be remarked here that the pulsation in the second left intercostal space is never observed except in cases which present so much dilatation as to allow of regurgitation at both auriculo-ventricular orifices. The diastolic murmur has been

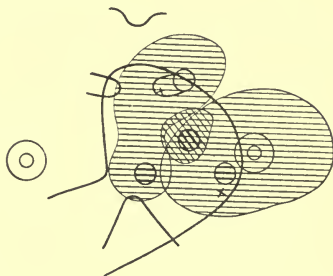


FIG. 171.—Distribution of murmurs in Case 48.

fully discussed in the chapter referred to. The systolic murmur heard over the sternal region of the præcordia appeared to be the same throughout, with two points of maximum intensity, and seems to me easily explained in this way, that, while at the lower end of the sternum it is heard with great distinctness, owing to the proximity of the muscular wall of the right ventricle and of the tricuspid valve, it is also heard with at least as much intensity over the conus arteriosus. It appears to me, in short, to be purely a murmur of regurgitation at the tricuspid orifice. While Russell's view as to the causation of the systolic pulsation in the second left intercostal space is to my mind absolutely proved, the murmur heard in that situation in the heart in debility seems to me to be simply a tricuspid systolic murmur propagated upwards by means of the conus arteriosus. It is quite analogous to the murmur produced at the right side of the heart in cases of heart strain, which is undeniably of tricuspid origin. To show that this murmur may have its greatest loudness close to the spot commonly known as the pulmonary area, the following case may be brought forward:—

CASE 48. *Cardiac Weakness from Alcohol*.—Sylvester N., æt. 24, unmarried, strapper in a stable, was admitted to Ward 6 of the Royal Infirmary on 26th June 1893, with obvious symptoms of alcoholism. His father, æt. 61, and mother, æt. 60, were in excellent health. Of ten brothers and sisters, only two brothers and one sister were alive, seven having died in their infancy. His social conditions were fairly good, except at times from his own fault. The patient's previous health had been quite good, but he had been much addicted to drink. The attack for which he was brought to the hospital began about Christmas 1892, since which time he had been drinking very heavily; about the middle of May pains in the legs, with some swelling of the ankles, set in.

On admission the patient was found to have great thirst and little appetite; the tongue was furred and shaky; the breath heavy and foul. No other symptoms connected with the alimentary or hæmopoietic systems were present.

There was breathlessness on exertion, and swelling of the ankles and legs. The pulse was of low pressure, moderate fullness, and perfect regularity. The rate was usually from 80 to 90. There was a well-marked venous pulsation in the neck. On inspection of the præcordia no impulse could be seen, and the apex beat could only be felt when the patient was placed on his left side. The deep cardiac dulness extended  $2\frac{3}{4}$  in. to the right and  $4\frac{1}{2}$  in. to the left of the middle line at the level of the fourth

cartilage. On auscultating the heart a soft systolic murmur was heard over a great part of the præcordia, with its maximum intensity over the left half of the sternum opposite the attachment of the third cartilage, as is shown in Fig. 172. It was obviously a murmur of tricuspid regurgitation, heard most distinctly over the infundibulum.

No abnormal symptoms connected with the respiratory or urinary systems were present. The patient had some insomnia, followed by restless slumber with alarming dreams, and he had a distinct tremor throughout the entire muscular system.

Under appropriate treatment the nervous disturbances passed away, and the patient was transferred to Ward 22, where, under the influence of cardiac tonics, he speedily lost all the swelling and breathlessness. The physical signs connected with the heart had in great part disappeared when he was discharged.

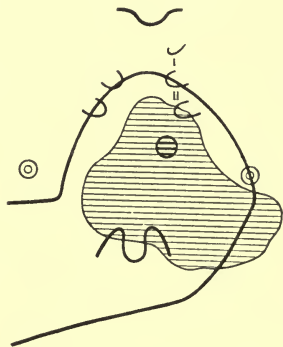


FIG. 172.—Cardiac dulness and murmur in Case 40.

In this case there could be no doubt that the murmur described was due to escape at the right auriculo-ventricular orifice, and its localisation throws much light on the question that has been discussed.

## ATROPHY.

A number of atrophic processes may be traced in the heart, which present a somewhat indefinite group of affections. It is beyond the sphere of this volume to discuss some of the conditions commonly termed atrophic, as for instance fibrillary, fragmentary, and pigmentary changes; with the exception, therefore, of retrogressive changes accompanied by alterations in the pigment, this section will be devoted to simple atrophy of the heart, or, in other words, cardiac emaciation.

The condition of cardiac atrophy, or a diminution in the size and weight of the heart, produced by lessening of the amount of its muscular tissue, has been recognised since the birth of morbid anatomy. It was, so far as is at present known, first scientifically observed by Riolan, and it was fully

described by Senac. Burns, Bertin, and Bouillaud gave in succession capital descriptions of the condition, and it has been generally recognised by all subsequent writers.

ETIOLOGY.—There are apparently some instances of small sized hearts of congenital origin, as was first observed by Burns. No hereditary influences, so far as has been discovered, are at work in the production of this condition. In such cases, the body has been otherwise well developed, but in other instances there has been a retention in adult life of some childish characteristics, so that the condition may have been part of what the modern French writers term infantilism. It is right to add that Parrot does not believe that such examples are really congenital; he holds that they are caused by a simultaneous arrest of the development of the heart and all other organs at puberty.

The common cause of simple atrophy of the heart is some wasting disease, such as, amongst many others, cancer, tuberculosis, syphilis, and diabetes. The statistics of Quain are probably the most important upon this subject, and may be consulted.

It is sometimes said that interference with the coronary arteries produces simple atrophy; but there will be abundant opportunity to show that they are more likely to give rise to degenerative changes. It has also been said that pericardial lesions, by producing compression of the heart, sometimes cause atrophy. Chevers appears to have been the first to suggest such a connection. As has already been shown, and as will be again referred to, there is more likelihood of hypertrophic than atrophic effects, and Kennedy only found 5 instances of atrophy in 90 cases of simple pericardial adhesions. The presence of an excess of fatty deposit occasionally gives rise to some degree of myocardial wasting. This, however, is excessively rare. One of the best examples is mentioned by Wilks and Moxon.

MORBID ANATOMY.—It has been common since the days of Bouillaud to speak of concentric, simple, and eccentric atrophy, as has been the case in regard to hypertrophy. That which was termed concentric by Bouillaud, followed by Walshe, or simple atrophy, as it has been called by Hayden, is the variety



almost invariably met with. A simple reduction in weight, from thinness of the walls—the simple atrophy of Bouillaud—is certainly rare, while the excentric atrophy characterised by diminished weight of the heart, along with an increase in the size of its cavities, is to be regarded as dilatation, and will be dealt with in the section devoted to that subject.

The great feature of cardiac atrophy is loss in weight, but in almost every instance there is also a diminution in the size of the heart affecting every dimension. The weight of the heart may be reduced in an adult to a very few ounces; in his *Lumleian Lectures*, Quain refers to the case of a girl, aged fourteen years, whose heart only weighed 1 oz. 14 dr. The cause of death in this instance was phthisis. The great characteristics of the heart, in addition to those just mentioned, are the removal of almost all the fat from the surface of the heart, so that its outline is less rounded than usual, and the blood vessels are more distinctly seen. The muscular substance, as seen with the naked eye, may be apparently normal. Sometimes, however, it is paler in colour and softer in consistence than is natural; but, on the contrary, it is sometimes darker and tougher than in health. In the case of simple atrophy the colour is unchanged, but the consistence may be somewhat firmer, and if there be paleness or darkness of tint it means that there is some fatty or some pigmentary alteration; in other words, it is no condition of simple atrophy, but is on the one hand fatty degeneration, or on the other brown atrophy. On microscopic examination, in simple atrophy the muscular fibres are found to be smaller in size, and, according to Letulle, the muscular cells, in addition to being diminished in size, lose their cylindrical aspect, and have a tendency to fusiform outline. According to this author there is, further, an increase rather than a diminution in the transverse striation and longitudinal fibrillation.

The morbid conditions constituting cardiac atrophy may be attended by other changes, as, for instance, the general or local affections which have produced the change. To these it is unnecessary to devote any further attention. It has been noticed by Bamberger that the amount of pericardial fluid is increased, apparently as a consequence of the atrophy. From my own

observations, however, it seems to me that in most instances the pericardial sac contains rather less than more fluid.

When coloured tissues undergo atrophy or involution, there is almost invariably a concentration of the pigment. This is more particularly seen in the muscles, and especially in the myocardium, where it constitutes the well-known pigmentary atrophy. In the hearts of almost all above middle life there is some collection of the pigment around the nuclei, but

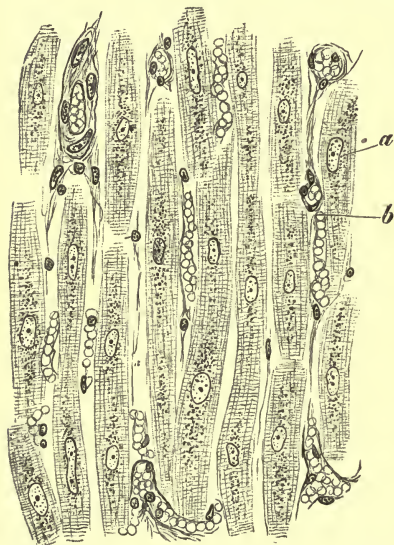


FIG. 173.—Pigmentary atrophy,  $\times 300$ . *a*, Muscle fibres showing concentration of pigment at the poles of the nuclei; *b*, vascular connective tissue between the fibres.

this does not exist to such an extent as to constitute the disease in question. Pigmentary atrophy is the result of a number of affections interfering with general nutrition. The heart in this lesion is small, hard, and tough. Its vessels are frequently somewhat tortuous. The section is of a chocolate tint. When examined under the microscope the fibres may be small; they retain their striæ, and around the nuclei are deposits of pigment forming fusiform collections which are composed of granular hæmatoidin.

There is often also some newly-formed fibrous tissue. The microscopic appearances are seen in Fig. 173.

**SYMPTOMS.**—The clinical features in cases of cardiac atrophy necessarily depend in great part upon the conditions under which it takes its origin. When it is produced, as is so commonly the case, by some malignant invasion of the digestive organs, it is possible to make out the appearances with considerable precision; but if the case should be a tuberculous affection of the lungs, certain of the appearances must be discounted as being the result

of alterations in the chest, produced by the pulmonary changes.

The general symptoms are, as a rule, simply those of the condition which has induced the atrophy; but it has to be remembered that atrophy following upon the primary condition accentuates some of the symptoms, as for example the general weakness from which the patient suffers. Debility and breathlessness are the most common complaints, but palpitation on exertion is not uncommon. There is not infrequently oedema of the dependent parts.

Physical examination shows, as a rule, empty arteries and low blood pressure. On inspection of the præcordia, the impulse may be weak or strong according to circumstances; all depends upon the condition of the chest. If there be retraction of the lungs, especially of the left lung, the apex beat may appear to be further to the left than normal, and may be more distinct than in health. The same is true in regard to the results obtained on palpation, since if the heart is uncovered, the apex beat may appear to be strong even when there is considerable atrophy and less energy. On percussion, the area of cardiac dulness is reduced. Here it must be noticed that there is a possibility of error, as in cases of emphysema the cardiac dulness sometimes appears to be very considerably reduced. It is probable that in such cases, from the increase in the breadth of the chest antero-posteriorly, the heart comes to lie more directly backwards and forwards, so that the area of dulness is in such conditions reduced. The cardiac sounds are, as a rule, diminished in intensity; but if the heart is close to the surface, as is often the case, the sounds may appear abnormally clear and ringing.

DIAGNOSIS.—Diminution in the area of cardiac dulness is the sole criterion during life by which cardiac atrophy may be determined; but in order to do this, the possibility of emphysema, or any other lung condition interfering with the area of dulness, must be eliminated. With care in this respect, the presence of diminished cardiac dulness, along with feeble pulse, breathlessness, and weakness, may be regarded as sufficient evidence, in the presence of some condition likely to give rise to atrophy, to warrant the diagnosis of the condition.



PROGNOSIS.—The prospects are entirely subsidiary to the primary condition which has given rise to the atrophy, and, as a rule, in this condition it is hopeless.

TREATMENT.—The aim in treating cases of cardiac atrophy must always be, if possible, to combat the conditions which have produced it, and seeing that these are for the most part fatal, but little can be done to remedy the cardiac condition.

### DEGENERATION.

There are several regressive processes, commonly classed as degenerations, by which the heart is affected. Fatty, amyloid, and hyaline degeneration are found in connection with the myocardium, but they occur in very different degrees of frequency. Fatty degeneration is very common, while waxy and hyaline changes are comparatively rare. In this section the abnormal conditions connected with the amount and distribution of fat will alone be considered, seeing that amyloid degeneration constitutes a mere pathological curiosity, and the hyaline transformation will be more fitly discussed in connection with another section.

### THE FATTY HEART.

Under the general term fatty heart are included not merely many degrees of change, but even absolutely distinct processes. There is, for instance, in the first place, simply accumulation of fat in the situations where it is usually found, more particularly in the epicardium. There is, secondly, infiltration of fat in situations which usually contain none, as for instance in the intermuscular tissues of the heart, or in the connective tissue of the endocardium. In the third place, there is true fatty degeneration, characterised by loss of the normal protoplasm of the cell and the formation of fat instead of it.

An excess of fat in the sub-endocardial tissues was observed by Harvey, Bonet, and Lancisi; it has been referred to by almost every subsequent writer. Senac sedulously investigated the amount of fat proper to different periods of



life, and Morgagni even more carefully investigated the condition from the pathological standpoint. Portal attempted to correlate the fatty overgrowth in the heart with an adipose condition of the skeletal muscles. Corvisart not only refers very fully to the condition of fatty overgrowth, but actually distinguished between it and true fatty metamorphosis. Fatty metamorphosis of the muscles concerned in voluntary movements was observed by Haller and Vicq d'Azyr, and Corvisart states that some pathologists, with whom he agreed, were of opinion that similar changes might be the explanation of some abnormal cardiac symptoms. Duncan and Cheyne must be allowed the credit of having observed changes in the heart which to all seeming were instances of fatty infiltration going on to degeneration. Laennec was undoubtedly the first to describe fully the characters of fatty degeneration, and to determine its identity with the processes discovered by Haller and Vicq d'Azyr; it is to him that we owe the name of fatty degeneration. Several other writers dealt with the subject immediately after the publication of Laennec's great work, to whom it is unnecessary particularly to refer. Rokitansky's investigation of the microscopic characters of fatty degeneration must, however, be mentioned, as it served as a point of new departure. He was followed by Peacock, Paget, and Virchow, while the entire subject of fatty diseases of the heart was most fully discussed in an elaborate memoir by Quain, whose treatment of the subject has been followed by most subsequent authors.

### FATTY INFILTRATION.

Accumulation of fat may be only transitory in consequence of some temporary disturbance of ordinary processes, or it may be permanent, from some lasting interference with tissue changes. In the former case it may almost be regarded as physiological, but in the latter it is pathological. Infiltration is almost invariably a sequel to accumulation, and it is to be regarded as an encroachment upon the healthy tissues by a deposit spreading inwards from positions where it is normally present.

ETIOLOGY.—The factors which lead to such changes are manifold. Hereditary tendency cannot be doubted, and fatty overgrowth of the heart is very commonly associated with a liability to general fat accumulation. It is probable that the male sex is more prone to such changes than the female. The statistics of Quain and Hayden are distinctly in favour of this view. Age is of even greater importance, the fatty overgrowth being rare in early life, and becoming more frequent until the middle and later portions are reached. It seems to be most common between the ages of fifty and sixty. Lack of exercise and want of fresh air are also important agents. Food seems to have important effects, but it may be doubted whether it is so much the nature as the amount of food which plays the greater part. It is believed that starch, sugar, and fat in excessive quantity lead to the deposition of fat in the tissues. Nitrogenous food, however, is quite capable of rendering a considerable amount of fat to the tissues when metabolism is defective. The abuse of alcohol, more especially in the form of heavy malt liquors, is decidedly powerful in producing deposits of fat. Apparently certain abnormal conditions of some of the great viscera concerned in metabolism, more especially the liver, may give rise to accumulation and infiltration.

It is obvious that such changes must be due either to an excessive supply of material out of which fat may be formed, or to a diminution of the processes by means of which the fat after being formed is disintegrated and removed. This is, however, merely stating the facts without attempting any explanation of them. It is, in fact, impossible to arrive at any reasonable explanation of the process. Under precisely similar conditions in two individuals, one may show well-marked fatty accumulation and infiltration, while another seems to be absolutely exempt from any tendency of the kind.

MORBID ANATOMY.—The usual positions for fat in the healthy heart are in the grooves between the auricles and ventricles, and between the two ventricles; but from these the fat passes over the ventricles, more especially upon the right ventricle, and lies in the sub-epicardial tissue. An excessive amount of fat sometimes obscures the whole of the heart.

More commonly, however, some ruddy isles of normal tissue are seen in the midst of a yellow sea of fat. If the process goes on further, the fatty deposit encroaches upon the myocardium by spreading along the intermuscular textures, so as to reach the sub-endocardial tissue. In this latter case condition is properly termed infiltration. When the condition persists for any length of time, there is apt to be a degeneration of the muscular fibres in consequence of interference with their nutrition. This, however, must be considered in the section which follows.

**SYMPTOMS.**—The usual symptoms of which complaint is made are feelings of oppression and breathlessness on any exertion, as well as the consciousness of being easily fatigued by exercise, whether of brain or muscles. Faintness and giddiness are often experienced.

It may be taken as the common experience that the digestive processes in this affection are but little, if at all, disturbed; in fact the primary functions connected with digestion are in most cases only too good. The stomach is not uncommonly somewhat dilated from long-continued distension, and the liver is quite as frequently enlarged from an excessive supply of nutriment. The symptoms connected with the heart are usually the result of enlargement of the organ and enfeeblement of its action. There may, however, be considerable fatty deposit without any obvious increase in the size of the heart. One of the most usual symptoms is a cyanotic tendency on exertion, attendant upon the dyspnoea above referred to.

The general adipose condition of the subcutaneous textures renders inspection of the neck and chest, as a rule, negative. But sometimes, and this is more especially the case when fatty infiltration is combined with hypertrophy of the heart, there is a wide diffuse præcordial heaving, with some indefinite pulsatile movements of the neck. Palpation generally reveals feebleness of impulse, unless hypertrophy is also present. The heart is frequently of nearly normal size, but there is commonly some increase of dulness; this is usually from dilatation, but often from hypertrophy—rarely from fatty deposit alone. The heart sounds are feeble. Even in the presence

of hypertrophy they are low in tone and weak in intensity, partly from the adiposity of the parietes, but also from interference with the energy of the heart. The first sound suffers more than the second. It is by no means common to find murmurs, but the first sound may be replaced by systolic murmurs over both sides. The pulse presents many different varieties. The wall is sometimes rigid, the vessel full, and the pressure high; at other times these are respectively elastic, empty, and low. Different combinations may be produced by modifications of the factors concerned in the pulse. When there is any degenerative change from pressure on the fibres by accumulation and infiltration, a periodicity of the pulse, to be discussed in the sequel, may appear. There are sometimes different forms of irregularity. Periodic changes in the breathing, of the Cheyne-Stokes type, may be present. If there be failing power of the heart, pulmonary œdema may give its characteristic physical signs. In the same way there may be scanty urine and albuminuria, as well as œdema of the dependent parts. The nervous system is sluggish, and soporose or sleepless tendencies, with impaired memory, are often noticeable.

The disease is apt to be progressive unless measures are taken to avert its further development. It is one of the common causes of sudden death, and this may be brought about by syncope or by rupture. More usually there is a gradual failure of energy throughout the whole system.

DIAGNOSIS.—The determination of fatty infiltration may be attained, if, along with an adipose condition of body, there is evidence of weakness of the cardiac impulse and sounds.

PROGNOSIS.—The prognosis depends on general considerations regarding age and energy, but there are two special points of importance—family tendency and personal habits. Beyond a doubt, hereditary tendencies exercise a powerfully determining influence and should be taken into account, while the possibility of self-control figures almost as largely in an estimate of the future.

TREATMENT.—The general management of this form of fatty heart is based upon that of corpulence; it therefore essentially concerns itself with the regulation of the habits of



the patient. The food must be arranged so that, with adequate nutriment, there must be no excess. Quantity is of more importance than quality as regards food; at the same time, starch, sugar, and fat should not be allowed to bulk largely in the diet. Exercise must be inculcated, both of body and mind. Attention should further be directed to the functions of the skin and bowels, and both these eliminating channels must be kept in an active state. In many cases treatment by baths and exercises is highly beneficial, but it must only be the prelude to a thorough rearrangement of every habit. The treatment of those cases in which infiltration has led to degeneration will be discussed under the next head.

### FATTY DEGENERATION.

The second form of fatty heart, taking its origin in structural alterations of the muscle cell, now demands attention.

ETIOLOGY.—The causes which produce fatty metamorphosis of the cardiac muscle may very naturally be arranged in the groups of predisposing and exciting. The predisposing causes are by no means of equal importance as compared with those to be regarded as exciting, and yet they must not be altogether neglected. It is, however, to the exciting causes that fatty degeneration in the overwhelming proportion of cases is to be solely attributed.

Amongst the predisposing causes heredity has an important place. There is an inherited tendency to fatty degeneration, so strong that it almost seems as if many persons were destined from birth to such structural alterations. It is well known that sudden death is common in certain families, and, amongst the lesions which underlie the occurrence of such abrupt terminations to life, fatty degeneration is one of the most important. Men are certainly more liable to this degeneration than women. According to Quain the proportion is about four to one, but the previous observations of Ormerod, and the later statistics of Hayden give respectively three to one, and two to one. As regards age, while every period of life has exhibited examples of fatty degeneration, from the child of two, mentioned by Kerkerling,

onwards, it is far more common in middle age and in elderly people, and in them it appears to be but one aspect of a general senile change. The general habits have some influence, but this is by no means so well marked as has occasionally been held; still, sedentary habits lead, as will be seen below, to a tendency in this direction, and it will be also mentioned in the sequel that the abuse of alcohol is an important factor.

The exciting causes of fatty degeneration are associated with alterations (1) in the quality of the blood, (2) the blood supply, and (3) the amount of work which the heart muscle is called upon to perform.

The condition of the blood may be modified by a number of different disturbances, many of which are chronic and long-continued, while some are acute and rapid in producing their effects. Any condition of cachexia may give rise to fatty degeneration, as was apparently first noticed by Ormerod. Instances are most common in such diseases as long-continued suppuration and phthisis, but are by no means uncommon in cancer and sarcoma. Inanition may likewise lead to it in the absence of any destructive lesion in any part of the body. Anæmia of every kind may be followed by fatty degeneration of the heart. It has been produced experimentally by repeated blood-letting in the lower animals, and it occurs in every variety of anæmia in the human race. It is, however, relatively rare in chlorosis, while extremely common in pernicious anæmia. In leucocythæmia it is almost as frequently met with as in this form of anæmia. It occurs as one of the results of diabetes, and here it may possibly be the result of some form of intoxication, produced by decomposition of the products circulating in the blood. Scurvy, purpura, and hæmophilia have all been found linked with the change. Chronic gout may produce fatty degeneration, but it is probable that this result is brought about not so much through the direct influence of retained waste products in the circulation as by the undeniable tendency to interference with the lumen of the blood vessels in the arthritic diathesis, as chronic renal disease is not infrequently found to be associated with it. It is probable that the metamorphosis

has its origin in retention of effete products within the blood vessels. A very large number of acute diseases give rise to fatty degeneration by acting through the blood. The association of fatty degeneration in acute diseases was observed, in the first place, by Laennec, but it is more particularly to Louis and Stokes that we owe our early knowledge of the changes in the myocardium which take place in acute diseases. It is found in typhus fever, enteric fever, erysipelas, diphtheria, smallpox, and septicæmia, as well as in many tropical diseases. In pneumonia it has often been found, and the tendency to this complication is undoubtedly one of the reasons for the great liability to cardiac failure so often seen in the course of this disease. Arising in the course of some of these affections, the myocardial change may be regarded as an infection; that is to say, the organisms actually circulate in the blood, and thus produce the destructive alterations. In some cases the process is to be regarded as an intoxication, and the products only of bacterial activity circulate in the blood. Certain poisons introduced into the system from without have a powerful effect upon the myocardium. The one which is most commonly encountered is naturally alcohol, although it is probably not by any means that which is most potent. This substance has been proved to increase the quantity of fat in the blood to a notable extent. Phosphorus is probably the most active substance as a cause of fatty degeneration. Within a very few days after the administration of phosphorus, even in a small quantity, fatty degeneration sets in, and affects the heart as well as almost all the internal organs.

Alterations in the amount of blood are believed to produce fatty degeneration. The change has been repeatedly seen after severe losses of blood, and the experiments of Perls have shown that frequent blood-letting in the dog can produce the structural alterations now under discussion. In all such conditions the parts of the heart which suffer to the greatest extent are the papillary muscles. In conditions of symptomatic anæmia there must be a hydræmic tendency, as the quantity of the blood is very speedily brought back to the normal by the absorption of lymph from the tissues. Venous engorgement was brought into prominence by Jenner, as a not

unimportant factor. It will be seen afterwards as one of the causes of fibrosis, and it is probable that while it may give rise either to fibroid or fatty changes, according to the individual peculiarities of the case, changes in the coronary vessels are not uncommonly followed by some fatty degeneration. The terminal arrangement of the blood vessels, already referred to in previous sections, which was first observed by Swan, and applied to the explanation of cardiac changes by Quain, renders it impossible for any deficient supply to be compensated by encroachment from a neighbouring zone. Here, as in the case of venous engorgement, precisely similar changes in the coronary vessels may produce in one individual fibroid effects, and in another fatty degeneration. Fatty overgrowth, when encroaching upon the muscular tissue, has an undeniable tendency to produce degeneration in it, and hence it is extremely common to find in the later stages of fatty infiltration that degeneration is associated with it. Exactly the same effect is found in cardiac hypertrophy, in which, when there is any considerable increase in the muscular tissue of the heart, a well-marked fatty degeneration occurs. This is, indeed, such a noteworthy fact as to lead some observers to assert that fatty degeneration is always associated with hypertrophy; and one of the most recent writers upon this subject has gone so far as to deny the existence of a compensatory hypertrophy altogether. The pressure of new formations, and more particularly of gumma, may give rise to fatty changes, but it is possible that in the latter case the specific infection has something to do with the change.

It is a well-known fact that the ordinary skeletal muscles undergo fatty degeneration from disuse. It is impossible, however, to believe that such a state of matters can occur in regard to the heart, seeing that it is always in action; but although absolute disuse is out of the question, lessened activity may indirectly give rise to fatty tendency. The amount of energy displayed by the heart is proportional to the general activities of the body, and when these are lessened the heart must necessarily have diminished exertion, while all the metabolic functions are throughout conducted on a low level. The heart itself does not receive sufficient stimulus, and its



nutrition may reasonably be expected to be less than under ordinary circumstances.

Whether any process comparable to the degeneration of an ordinary skeletal muscle after it has been separated from its proximal centre ever occurs with respect to the heart, has never been determined. It is, however, within the region of speculative possibility that from certain structural changes in the *vagus* some alterations may occur in the muscles.

**MORBID ANATOMY.**—The appearances arising in consequence of this form of degeneration are far from uniform, and differ chiefly according as the change is general or local. The heart is not increased in size in pure fatty degeneration, but as fatty degeneration frequently accompanies or succeeds conditions of dilatation and hypertrophy, it is common to find large hearts showing every appearance of this degenerative change. It occasionally happens that the heart may look larger on account of dilatation, which has followed upon the degeneration, and it has often been remarked that the organ looks enlarged even when its size is normal, simply on account of its want of consistence, which produces a tendency to flatten itself out, and therefore cover a larger area when placed on any object. Instead of being enlarged, the heart in fatty degeneration is often found to be diminished in size. This occurs more particularly when the degeneration is the result of some long-continued chronic disease.

When taking origin in acute disease the process is usually a widespread one affecting almost the whole heart, although even in such cases the effects are more obvious in the left than in the right ventricle. The muscular tissue of the heart is commonly dark in colour, on account of staining from blood destruction. In these instances it may not at first be very apparent that fatty degeneration is present; even under such circumstances, however, there is often, at least in parts, some diminution in tint.

In other cases arising in consequence of chronic affections, such as profound anæmia, the colour of the whole heart is paler than usual; but there are usually areas in which the pallor is more conspicuously seen. Even in acute diseases the heart is sometimes pale instead of deep in colour, and it was

described by Laennec as that of a dead leaf. This is the case more particularly as regards the papillary muscles and the sub-endocardial tissues. In degeneration arising from such chronic causes as local obstruction of the blood supply, the change is purely local. It is much more common in the left ventricle than in the right. In such local manifestations the result is invariably to produce an area characterised by paleness. No matter where or how widespread the changes, they are accompanied by alterations in the consistence of the part which is soft and easily torn, as well as friable. The resistance of the tissue is so greatly reduced that there can be no doubt it may be torn by energetic systole of the heart. It has also a feeling of greasiness, and, although the fact has been doubted, there is an increase in the amount of fat in the tissues. According to the researches of Böttcher and Krylow, the amount is increased from something like 2 or 3 to 4 or 5 per cent.

As has been said, the left ventricle usually suffers more than the right, and it will be well, before leaving the subject of the naked-eye appearances of such a heart, to observe that in all general fatty degeneration the left is more affected than the right ventricle, if it be not, indeed, solely implicated. From his investigations upon the subject, Quain came to the conclusion that in about half of the cases both the ventricles are affected, and that when only one ventricle is diseased, the left is affected twice as often as the right.

The auricles suffer much less than the ventricles. The observations of Ormerod led him to be sceptical as to the implication of the auricles, but there can be no doubt that they occasionally suffer. It is necessary in every example of structural alteration of the heart to study the condition of the coronary arteries. They are found to be affected in most of the cases of localised fatty change. The most common change in the coronary vessels is arterial sclerosis, often with some calcification in the walls, as well as some thrombosis within the lumen of the vessels. There is also, however, in localised fatty changes, embolism, which has nothing to do with, and usually occurs without, arterial sclerosis.

Fatty degeneration may be found in other organs, as in

the cells of the liver, the epithelium of the kidney, and the intima of many of the arteries. All these changes are closely linked with the cardiac alterations, and own the same causes.

There are, moreover, alterations in other organs consecutive to the cardiac changes. These are due to want of energy. Amongst such effects must be mentioned venous stasis of the dependent parts, and its consequences. It is unnecessary to pass any view on the different modifications of tissue which result in this way; suffice it to say that there is no means of distinguishing between such changes and those which take place as the result of cardiac failure from valvular disease, especially affecting the mitral cusps.

The microscope reveals the existence of fatty degeneration when it is in such an early stage as to be absolutely unrecognisable by the unaided eye. The first appearance which is to be detected consists in the presence of a small number of fine granules within the muscular fibres. They are usually arranged in a longitudinal manner, parallel to, and perhaps between, the fibrils, but are occasionally irregularly scattered through the fibres. In such an early phase there is little, if any, change in the transverse striation, and the sole alteration, therefore, is restricted to the existence of the granules. These can be best seen in sections stained with osmic acid.

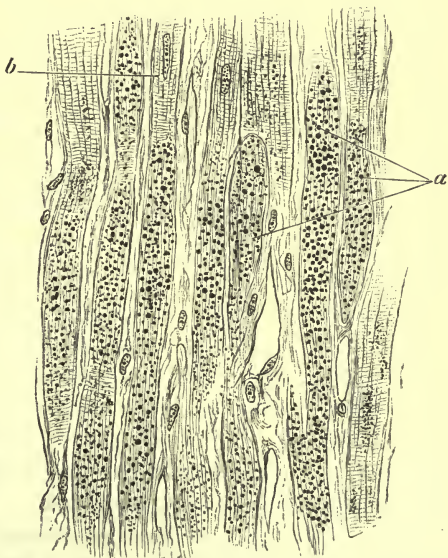


FIG. 174.—Section of myocardium from a case of pernicious anæmia showing fatty degeneration,  $\times 250$ . Osmic acid. *a*, Fibres with numerous oil-droplets; *b*, fibres unaffected by the change.

In later stages there is an increase in the number of the granules, with a corresponding loss of striation, and the nuclei



in the affected fibres commonly disappear. There are usually some portions of the section under examination in which fibres may be observed to be free from granules but deprived of striæ, so as to present an appearance nearly homogeneous, or only marked by the longitudinal fibrillation. All these changes may be seen in Fig. 174, which shows the early phase, with granules and striæ, and the later, in which the granules have increased and the striæ disappeared. There are also at one or two points some almost homogeneous portions of fibres.

In still more advanced conditions the granules become larger and yield the appearance of globules, with a translucent aspect. They vary much in size, but are not often larger than a blood corpuscle. The longitudinal arrangement is still preserved as a general rule, but there are many exceptions, in which an indiscriminate distribution can be seen. Some of the fibres are seen to contain nothing but such globules, but more commonly some portions of even the most affected fibres have comparative freedom. One point is clear. In their localisation the globules constantly accumulate, as Gowers insists, between the primitive bundles, but it has not been, and probably cannot be, determined whether this is due to coalescence of granules formed there, or to passage outwards from the fibres.

Just as there is a strong tendency for the selection of certain spots as the principal points of attack by this form of metamorphosis as seen by the naked eye, so under the microscope there is a singular grouping of the affected fibres—of two adjacent fibres one may be healthy and its neighbour diseased, nay, one small part of a fibre may be hopelessly destroyed, while the rest is sound.

The nature of the process resulting in fatty degeneration is at present absolutely obscure. Fat exists in every animal tissue, but in such a state of fine division as to be invisible under the microscope, and to be recognisable only by chemical analysis. The amount contained in the heart is from 2 to 3 per cent. of the total weight of that organ. When moderate fatty degeneration is present, so that the granules and globules may be seen in the muscular fibres under the microscope,



there is, according to Ormerod, no increase in the absolute quantity of fat, and it therefore seems that the first step of the degeneration, as Gowers says, is a separation and precipitation of the fat previously combined. Possibly some of the granules first seen as above described, in the process of degeneration, are not all composed of fat, and it is to be remembered that Virchow suggested that albuminoids may become altered into some soluble extractive and be absorbed, leaving behind the fat in a state of precipitation. When the change proceeds further, the amount of fat is larger than can be explained in this way, since the amount of fat, as above mentioned, may be double that normally present. One explanation is, that it is produced by a metamorphosis, a chemical process, in which, by impaired tissue change, there is a destruction of the fibre. The fact is undoubted that fat may be formed from nitrogenous substances. The formation of adipocere has been cited by Quain in illustration of this fact, and he himself showed that by prolonged maceration in dilute acetic acid, healthy muscular fibres undergo fatty degeneration. As an explanation of the possibilities by means of which such metamorphosis may take place, Rindfleisch has shown that in cloudy swelling the granules in the cells are soluble at first in acetic acid, but that this condition passes into another in which the granules do not dissolve in that acid, but are readily soluble in ether. The fact that regions from which the blood supply is altogether cut off, by embolism or thrombosis, undergo fatty degeneration, is sufficient to prove that such changes may be entirely due to alterations going on within the tissues.

It must be admitted, however, that in addition to such alterations, some of the fat may be brought from without. Robin and Verdeil, Ormerod, and Walshe believed, indeed, that all the fat seen in the muscular fibres was introduced from without. This is extremely improbable in such instances as the acute fatty degeneration of the febrile state, and it is impossible in those localised patches of degeneration which occur as a consequence of vascular occlusion. It must, nevertheless, be allowed that in a very large number of cases showing fatty changes both processes may be at work.

SYMPTOMS. — The clinical features produced by fatty degeneration are characterised by indefiniteness. This arises not only from the fact that the changes in the muscular wall have their origin in a good many different conditions which in themselves give rise to disturbances of the circulation, but also from the circumstance that the various appearances yielded by fatty degeneration may have their origin in other conditions of weakness.

The most common complaints made by patients suffering from this affection are breathlessness, especially on exertion, and debility, manifested on attempts to employ the muscles. There is also, in some instances, a degree of uneasiness, or even pain, in connection with the chest, while interferences with the functions of the brain, as regards sleep, memory, and intellectual processes, are not uncommon.

The general appearance in fatty degeneration is so dependent upon the conditions under which it arises, that no general statement can be hazarded. It is customary to say that the face is usually pale, but this is only true of certain cases, and well-marked fatty alterations may be present in those who are ruddy. Between the appearance of the face in cases of fatty degeneration in pernicious anæmia, and those attendant upon a similar change in the course of chronic alcoholism, there is every gradation. It must, however, be admitted that the chronic alcoholic patient most liable to fatty degeneration is more likely to manifest a waxy pallor than a ruddy glow. Senile patients in whom, from free habits of living and inefficient exercise, fatty infiltration has passed on to degeneration, have frequently a mixture of ruddiness and pallor, along with yellowness of the conjunctivæ from adipose tissue, and the arcus senilis, which has been proved to be the result of fatty degeneration.

Sometimes there are evidences of œdema, more especially in the dependent parts, but this is not, as a rule, great, except in those cases which have led to considerable cardiac dilatation. It is rather common, on the whole, to find a tendency to chilliness of the extremities. In chronic forms the temperature is usually subnormal.

The digestive functions are, as a rule, impaired. There

is commonly deficient appetite and sluggishness in the performance of all the various functions concerned in digestion, and in addition, there is not infrequently gastro-enteric catarrh. Needless to add, in the type of fatty degeneration resulting from infiltration, it is by no means uncommon to find some enlargement of the liver.

The condition of the blood depends entirely upon the conditions which have led to the degeneration. As tested by the usual clinical methods, there may be deficiency of the hæmoglobin, hæmocytes, and leucocytes, as in cases of profound anæmia; or, as in other instances in which cardiac dilatation has ensued upon chronic degenerative processes, there may be, as the result of cyanosis, an increase in all.

The more special symptoms connected with the circulatory apparatus are, like those which have been considered, subject to considerable variation. Uneasy sensations, often amounting to actual pain, may be felt in the præcordia, and may radiate upwards and outwards to either one or other shoulder and arm, or may even implicate both sides. There are frequently sensations of disordered movement within the chest, such as tremor cordis. Syncopal attacks, sometimes producing absolute unconsciousness, often occur, and overshadowing all these symptoms there may be the awful sense of approaching death. These subjective sensations present a very close resemblance to angina pectoris; in fact, it would be better to say that in such instances angina pectoris in its varied manifestations constitutes a feature of fatty degeneration.

The pulse almost invariably reveals some well-marked changes. The arterial wall may be soft and yielding when the affection has had origin in some acute disease or anæmic condition, but it is, on the other hand, rigid and inelastic in many of the senile forms of the disease. The pressure is almost invariably diminished, but this is sometimes rather difficult to estimate in the forms marked by arterial sclerosis. The vessel is almost invariably rather empty; the rate of the pulse presents great differences. In the more acute forms the rate is usually accelerated, so that the pulse may be persistently more than 100 per minute. In the more chronic forms, especially those which are senile, the rate of the pulse

is commonly diminished, and for long periods of time it is possible to determine that the rate is never above 40, and sinks to 20, or even less. The rhythm may also present different characters. In some instances it is perfectly regular, excepting when a transitory tumultuous action comes on, but many different changes in rhythm may be present. Simple irregularity is common, but a grouping of the pulsations is by no means infrequent. One feature has on several occasions attracted my attention: a singular periodicity of the pulsations has occurred both as regards the rate and the fulness of the pulse, so as to give appearances which might be regarded as somewhat analogous to the Traube-Hering curve, revealed by physiological experiments in the case of the arteries. Over and above such appearances, there may be a distinct loss of some of the heart beats in the passage of the blood wave to the periphery, so that the number of pulsations, as observed in the radial artery, may be smaller than the number of cardiac contractions.

The neck and præcordia rarely give any noteworthy appearance, with the exception of absence of all apparent impulse. The apex beat may be perfectly invisible, and no impulses may be seen in the lower sternal region, in the jugular fossa, or in the neck. The application of the hand to the præcordia largely confirms this negative evidence, since the apex beat is commonly found to be extremely feeble. It usually occupies a situation very near the normal position, and it gives the feeling of a short feeble tap to the hand. Even this may not be perceptible, when the only impulse to be felt is at the lower end of the sternum at its left edge. This observation was originally made by Stokes, to whom we owe so much in the study of cardiac degenerations. The area of cardiac dulness is not, as a general rule, much altered, unless some degree of dilatation is present, in which case the extent of outline conforms to that which will be described under that head; but it must be remembered that in those cases of fatty degeneration which have taken their origin in, or have been associated with, hypertrophy, the area of cardiac dulness may be considerably augmented. The heart sounds invariably undergo considerable change. The most common experience is to find



such a degree of enfeeblement of the first sound as to render it less audible than the second, and sometimes, as was pointed out by Stokes, on listening at the apex, only the second sound may be heard. In most cases, at the lower end of the sternum the first sound is present, although it may be extremely weak. When there is acceleration of the rate of pulsation, the heart sounds, from equidistance and equal loudness, may assume a foetal character. Systolic murmurs may replace the first sound over both the left and the right ventricles, more particularly in cases arising from febrile disturbance or profound anæmia.

The respiratory system almost invariably yields distinct symptoms on investigation. Dyspnœa on exertion is at once an early and persistent accompaniment of the condition, but in later stages constant anhelation may be present, and the breathing has often a suspirious character. The rhythm often undergoes alterations so as to present periodic appearances. Cheyne-Stokes' respiration is occasionally present. Stokes believed that this ascending and descending respiration was pathognomonic of fatty degeneration, but, as has been shown previously, this was soon found to be a baseless supposition. Certain of the modified respiratory movements are common in fatty degeneration. A persistent tendency to sighing, or irresistible inclination to yawning, may be mentioned amongst these.

The renal secretion is almost always scanty, high coloured, and of increased specific gravity. Not infrequently it contains albumin and tube casts.

The painful sensations experienced in the region of the præcordia have already been referred to, and require no further comment. Feelings of faintness and giddiness are common. The functions of the brain are often interfered with, more particularly as regards sleep. A persistent soporose tendency is highly characteristic of fatty degeneration, and even after having slept for the greater part of twenty-four hours, the patient on being awaked may complain of being unrefreshed. But, on the other hand, insomnia is occasionally found, or—and this has been frequent in my experience—the nights have been passed in fruitless endeavours to fall asleep, while the

daytime has been spent in a vain struggle to overcome an overwhelming desire to sleep. A gradual alteration in the higher mental processes may often be determined; the memory, more particularly, suffers, but all the mental operations become blunted, and the temper is at the same time apt to undergo changes, usually for the worse. The brain powers appear to be easily exhausted by even slight mental exertion. In addition to such persistent appearances there are often others of a more transient character. Attacks of an apoplectiform or epileptiform nature are sometimes observed, the latter presenting close resemblance to *petit mal*. Transitory unconsciousness and passing aphasia are the most common appearances to be classed under these heads.

The course of the disease is marked by the same variability as obtains in the evolution of the symptoms. In the slowly developed forms of the affection, arising from accumulation and infiltration, the duration is lengthy, but in the rapidly produced lesions of acute diseases and profound anæmia the course is much less prolonged. The termination is, in most instances, brought about by heart failure, gradual or rapid, according to circumstances. It is in this cardiac disease above all others that sudden death from asystole most commonly takes place. An abrupt termination to life in aortic incompetence is most commonly associated with this degeneration, so often attendant upon the inevitable hypertrophy arising from that valvular lesion. This manner of death is not, however, the only startling close of life. Rupture of the heart, although perhaps more common in fibroid changes, takes place in fatty degeneration also, and furnishes another example of sudden death.

DIAGNOSIS.—There can be no question that Balfour is correct in doubting the possibility of absolutely diagnosing a fatty heart. From the different symptoms, general and local, there may be a reasonable probability of the existence of this degeneration, but the diagnosis cannot, except in very rare cases, pass from the region of inference to that of certainty.

The clinical features directly connected with the circulation are solely those of loss of heart energy—feeble cardiac impulse, weak heart sounds, and small arterial pulsation, with

cyanosis and dyspnoea in most instances. These appearances have nothing characteristic in themselves. When accompanied by painful sensations and syncopal attacks, there will necessarily be some suspicion of myocardial change, and if there be at the same time any impairment of the cerebral functions, a reasonable presumption in favour of fatty degeneration may arise. Each and all of these features may be found in chronic myocarditis, and the differentiation of the two lesions is rarely possible.

Stress has been laid by many writers upon the existence of similar changes in other regions, as lending weight to the supposition of fatty degeneration of the heart. Evidences of arterial degeneration are often mentioned as confirmatory, more especially by Gowers, but it seems to me that such changes are always more in favour of the diagnosis of cardiac fibrosis, and that they cannot be allowed much weight in the diagnosis of fatty disease. The arcus senilis has, since Quain's observations, been accepted as furnishing an important symptom. While in itself of no moment, it may, along with the general features of a failing heart, be allowed to contribute slightly towards the probabilities in favour of fatty heart.

When a valid cause of degeneration is present it will materially help the diagnosis. It is unnecessary to say that a failing heart in acute disease, profound anæmia, and analogous conditions, is most probably in any case determined by fatty changes; this goes without saying. But where chronic heart failure occurs, without previous valvular disease, in any one who is of alcoholic tendencies, there will naturally be an inclination towards the diagnosis of fatty degeneration. In a similar way any one with a full habit of body and a large heart, who gradually develops the features of cardiac failure, will reasonably be judged to have degeneration as the result of infiltration.

PROGNOSIS.—Any attempt to form a forecast of the probable future of fatty degeneration must be largely based upon the causes which have led to the myocardial change. The prognosis, therefore, is very different in those cases which have their origin in acute, and in those arising from chronic disease. The acute form of degeneration constitutes the danger so

much dreaded in the course of the continued fevers, diphtheria, pneumonia, and similar diseases. When it shows itself by those features of cardiac depression which are so unmistakable, an estimate of the outlook will probably be based upon the period of the disease at which the change shows itself, and the vitality and energy of the patient. Speaking generally, if the cardiac weakness is manifested at an early stage of the disease, the forecast will necessarily be more serious than when it occurs at a later period. Similarly, in the weak and debilitated the prognosis is more serious than in those who are robust and healthy. As Gowers has so well put it, acute forms of degeneration are "those in which the immediate danger is greatest, but at the same time, the ultimate prognosis is usually favourable."

In the forms of degeneration occurring as the result of chronic disease, the immediate danger is not so great, but the ultimate prognosis is more serious. Here the whole future depends upon the possibility of removing the cause operative in the production of the affection. It is unnecessary to refer to prognosis in degeneration taking its origin in some incurable malady, such as malignant invasion of the system, or when the changes arise in consequence of senile degeneration, in which the conditions are, for the most part, unalterable; but in some forms of fatty degeneration caused by blood changes, toxic processes, and hygienic errors, the prognosis is more hopeful. In many forms of anæmia the primary disease may be entirely removed, and when this is the case the result may be perfectly satisfactory. In the same way, alcoholic fatty degeneration may be removed on cutting short the prime causal factor. Similarly, fatty degeneration following upon fatty infiltration may be averted by the adoption of such thorough-going treatment as will remove the deposit.

TREATMENT.—The management of the cases of fatty degeneration from acute processes, and from chronic disturbances, differs entirely. In the former case absolute rest, appropriate food, and diffusible stimulants, are above all else necessary. It is usually advisable to give the patient such cardiac tonics as digitalis or strophanthus, which may be most advantageously combined with strychnine, ammonia, and ether. The inhala-



tion of oxygen is often of the greatest benefit, and may cause a recovery when all other means seem likely to fail.

If the blood should be in an impoverished condition, the use of iron or arsenic, or both drugs, must be employed. The views of Hunter, as has been stated by me elsewhere, seem so reasonable as to merit a careful trial, and in my own hands the use of internal antiseptics in profound anæmia has certainly been beneficial, as elsewhere stated. In cases arising from toxic causes, no treatment is of any avail unless these causes are withdrawn, and in all chronic forms the observer would do well to inquire most diligently as to the possibility of alcoholic abuse. In those instances resulting from fatty accumulation and infiltration the treatment consists in the careful regulation of the diet and the inculcation of exercise.

In all cases of chronic fatty degeneration the habits, surroundings, and occupation of the patient may be watched. In some forms of fatty degeneration the method of treatment carried on at Nauheim is most advantageous, and a course of baths, accompanied by resistance exercises, and followed by carefully arranged exercise, will be found of incalculable benefit.

In addition to all these considerations, attention must be given to the necessity in many cases of employing cardiac tonics and stimulants. If there be any tendency to increased peripheral resistance, such cardiac tonics should be combined with the nitrites, and more particularly with nitro-glycerin. In many of these cases the iodides are of even greater utility, and iodide of potassium combined with infusion of digitalis can be cordially recommended. If pain is a prominent symptom, such remedies will be found in most instances quite sufficient to curb it, but it is sometimes necessary to step in and administer nitrite of amyl, if there be apparently much arterial spasm; ether or chloroform may be required, while in certain cases it is absolutely necessary to give the patient a subcutaneous injection of morphine.

CASE 50. *Fatty Infiltration*.—W. H., aged 70, retired manufacturer, frequently consulted me during the last few years on account of some uneasiness in the back of the chest, and considerable breathlessness on exertion. His family history was satisfactory, but showed some liability

on one side to heart affections; his father, belonging to a healthy house, lost his life, at the age of 40, by a boating accident; his mother died at the age of 64 of cardiac failure. One brother died, aged 59, from cerebral disease, a second at 40, from "weak heart"; another was quite well; a sister died of congenital heart disease, another of heart disease, and the only other one had a weak heart. All his sons and daughters, four and two in number respectively, were healthy, but there was a tendency to cardiac weakness amongst them, shown by a liability to dilatation under any strain. The patient had, on the whole, been a healthy man, but about the age of 50 he had to take a long holiday on account of cardiac weakness, undoubtedly due to muscular debility. Since that date, he had engaged very actively in business.

The patient was 5 ft. 10 in. in height, weighing between 16 and 17 stone; he always presented a healthy appearance, with a slight purplish tint, however, about the lips. He frequently complained of coldness of the extremities, which sometimes awoke him during the night. The skin acted very freely, the least exertion giving rise to profuse perspiration. The alimentary system was only marked by a slight fur upon the tongue, and an enlargement of the liver, which extended from the fourth cartilage to a couple of inches below the costal margin, or 7 in. in all. The thyroid gland and spleen were of normal size. The blood was never examined. The pulse, usually 64 per minute, was full, large, regular, and equal, while the walls of the vessel were in no respect altered. On inspection of the neck and præcordia there was absolutely no impulse to be seen, but this could not be wondered at, since the parietes were so well clothed. A diffuse impulse could be felt in the fourth and fifth left intercostal spaces, at a point  $4\frac{1}{2}$  in. from mid-sternum. On percussion the cardiac dulness was found to extend  $2\frac{3}{4}$  in. to the right and  $5\frac{1}{4}$  in. to the left of the middle line at the level of the fourth cartilage. The cardiac dulness began at the level of the third left costal cartilage. The first sound both at the apex and in the tricuspid area was distinct and clear, but not so intense as the second at the base, which was considerably louder in the pulmonary than in the aortic area. No accompaniment was ever detected. On examination of the lungs there was constantly a slight impairment of the percussion sound at the bases behind; and on auscultation fine crepitation accompanying the inspiration was to be heard over both. The extent over which the crepitation could be heard varied at different times, but was usually confined to a zone, about 3 in. broad, along the inferior margin of each lung. When there was any slight pyrexial attack, such as even a simple coryza, the area over which the crepitation could be heard was distinctly increased, and it sometimes reached almost as high as the angle of the scapula. Attacks of subacute bronchitis were frequent, but there never was hydrothorax. The urinary system was never in the slightest degree implicated. The integumentary system, apart from the tendency to profuse perspiration, was always unaltered, and no trace of œdema was ever observed. The nervous system showed not the slightest deviation from healthy conditions. Some strophanthus and nux vomica, with regulation of diet, rest, and exercise, have always sufficed to restore equilibrium.

This may be taken as an excellent instance of fatty infiltration, possibly associated with some degeneration, but which may perfectly well be a simple example of accumulation. It is of interest to observe that there has never been the slightest tendency towards anginous seizures, or any discomfort excepting that dull pain in the back which is so often found in cases of passive hyperæmia of the bases of the lungs.

CASE 51. *Fatty Degeneration from Pernicious Anæmia.*—T. S., aged 36, steel grinder, was admitted to Ward 22 of the Royal Infirmary, 7th December 1892, complaining of weakness. The patient's father had died at the age of 45 of some malignant affection, and his mother at the age of 56 from some pelvic disorder. The family had consisted of six brothers and two sisters, of whom the former were all alive, but the two sisters had died from causes about which the patient knew nothing. His social conditions had been eminently satisfactory, and his conduct all that could be desired. His previous health had been in all respects good until about three years before admission, since which time he had been gradually becoming worse.

The general appearance of the patient was somewhat striking. He was a man of medium height, 5 ft. 7 in., weighing 11 st. 8½ lb. His hair and eyes were of a dark brown hue, forming a startling contrast to the tint of the face, which was of a pale straw colour. The lips were intensely pale, as was the interior of the mouth, and on turning down the lower eyelid, the mucous membrane lining it was also found to be extremely blanched. The patient complained of no symptoms connected with the digestive system, but the tongue was large, pale, and flabby, and the liver extended in the mammillary line from the fourth rib downwards for a distance of 8 in. None of the lymphatic glands were enlarged, and the thyroid was of normal size, but the spleen reached the anterior axillary line. On examination of the blood, it was found only to contain 40 per cent. of hæmoglobin, and 1,600,000 red blood corpuscles. The corpuscles showed a great variety of sizes and shapes; megalocytes and microcytes were present, as well as a large number of poikilocytes. The white blood corpuscles were practically normal in number. He suffered much from breathlessness and palpitation, especially on the least exertion. The pulse was somewhat increased in frequency, being usually 106 or 108 per minute. The artery was soft and elastic; the vessel was rather empty, and its pressure was low; the pulsation was perfectly regular and equal. The heart was somewhat enlarged, its borders being respectively 2½ in. and 4½ in. to the right and left of mid-sternum. On auscultation systolic murmurs were heard, one in the mitral area, and another up and down the sternum, with its maximum intensity between the tricuspid and pulmonary areas. On examination of the respiratory, urinary, and integumentary systems, no abnormal points could be elicited; but as regards the nervous system, examination of the retina showed that there

were a number of retinal hæmorrhages. These were more particularly seen in the fundus of the right eye, but were also present in the left. The erect image of the right eye showed one large dense hæmorrhage around the fovea centralis, while several smaller hæmorrhages were scattered about between that spot and the disc. A number of smaller patches of hæmorrhage were scattered widely throughout the fundus. The erect image of the left eye showed a few patches of hæmorrhage around the fovea, and some others in the upper part of the fundus, more especially in the angle formed by the two main branches of the retinal blood vessels.

There can be no doubt that this case was an example of pernicious anæmia of a somewhat pronounced type. He was accordingly treated from the first with  $\beta$ -naphthol, receiving 3 grs. in pill three times daily. At the same time he was confined absolutely to bed, and his food consisted largely of farinaceous material. Under this treatment he made somewhat rapid improvement with a few fluctuations, and by the 13th of March he stated that he felt perfectly well. His hæmoglobin had reached 55 per cent., and the red blood corpuscles numbered 3,360,000. The condition of the circulation was also greatly improved; the murmurs had almost disappeared, and the heart sounds were loud and distinct, while his complexion, instead of being marked by a pale straw tint, was clear and almost rosy. He was accordingly, on 13th March, sent to the Convalescent House. On the 30th March he reported himself, and it was then found that his hæmoglobin was 60 per cent., and the red blood corpuscles 4,000,000. On examination of the fundus of the eye it was seen that the retinal hæmorrhages had absolutely disappeared. The patient was therefore allowed to go home. In order to present a succinct statement of the improvement he underwent, the following table may be given, from which the condition of the blood will be seen :—

Date.	Corpuscles.	Hæmoglobin.
December 7 . . .	1,600,000	40 per cent.
"    9 . . .	2,000,000	30   "
"   21 . . .	2,100,000	18   "
"   22 . . .	1,600,000	18   "
January 1 . . .	1,100,000	18   "
"   20 . . .	1,800,000	20   "
"   28 . . .	2,000,000	30   "
February 2 . . .	2,120,000	38   "
"    5 . . .	2,400,000	40   "
"    9 . . .	1,800,000	40   "
"   11 . . .	2,000,000	46   "
"   13 . . .	2,700,000	46   "
"   16 . . .	2,080,000	42   "
"   21 . . .	2,100,000	46   "
"   24 . . .	2,144,000	54   "
"   27 . . .	2,720,000	58   "
March 3 . . .	2,640,000	60   "
"    7 . . .	2,560,000	52   "
"   10 . . .	2,664,000	56   "
"   13 . . .	3,360,000	55   "
"   30 . . .	4,000,000	60   "



All went well for a short time, but in a few weeks the patient returned in a grave condition, the corpuscles having suddenly fallen to 800,000, and the hæmoglobin to 16. In spite of every means employed, including transfusion of blood, he rapidly sank, and died.

The post-mortem examination was performed the day after death by Dr. Robert Muir, from whose report the following condensed account is taken.

The body was well nourished. There was extreme pallor of the whole surface, with slight œdema of the legs and dependent parts; well-marked rigidity and slight lividity were present.

There was diffuse ecchymosis on either side of the outer surface of the pericardium, and the sac contained 2 oz. of serum. The left pleura contained 20 oz. of serum. There were adhesions on the posterior part of the lung, and over the apex. The right pleura contained 16 oz. of serum, but had no adhesions.

The heart weighed 13 oz. There were some minute ecchymoses over the auricles. There was no diminution of the fat upon the surface of the heart. The aortic and pulmonary cusps were competent. The cone diameters of the orifices were as follows:—Aortic, 1; pulmonary, 1·2; mitral, 1·5; tricuspid, 1·95. There was very extensive fatty change in the myocardium, both in the left and right ventricles, but especially in the former. The endocardium showed a very fine yellow speckling. The valves were normal, there being only some early atheromatous change at the beginning of the aorta, and in the anterior cusp of the mitral valve. The left ventricle was 4 in. long, and  $\frac{5}{8}$  in. thick at the thickest part. It was therefore somewhat hypertrophied and dilated. The columnæ carneæ were thin.

The left lung weighed 1 lb. 8 oz., the right 1 lb. 12 oz. There was a large number of white fibrous nodules in the pleura, about the size of a pin's head, along the junction of the interlobular septa. These showed no signs of caseation. There were some nodules of a similar kind in the lung, but fewer in number. There were, further, some small caseous masses in the upper lobes, evidently of old standing. The lungs were anæmic, and posteriorly œdematous. Both lungs exhibited these conditions, and the right in addition had some miliary tubercles.

The peritoneum was healthy. The liver weighed 4 lb. 12 oz. It showed a number of small depressions with thickening of the capsule, which gave an irregular character to the surface, especially over the right lobe. There were no corresponding fibrous bands running in from these. The liver showed marked fatty degeneration, especially in the centre of the lobes, with pigmentation at the periphery. On the addition of hydrochloric acid and ferrocyanide of potassium, a distinct iron reaction was obtained. The pigment was apparently abundant, and a section of the organ had a peculiar chocolate-like colour. The spleen weighed 11½ oz. It was considerably enlarged, but a section seemed normal, and gave no iron reaction. The left kidney weighed 9½ oz., the right 8½ oz. The cortex was enlarged, and showed well-marked yellow and red mottling. Fatty degeneration was well marked in both. The stomach was congested

posteriorly, but otherwise healthy. The duodenum and first part of the jejunum showed diffuse hæmorrhages into the mucous membrane, and were deeply bile stained. The rest of the intestine was normal. The pancreas and suprarenal bodies were quite healthy.

The bone marrow in the shaft of the femur was completely transformed into the red variety, and there was some softening of the bone trabeculæ. On microscopic examination this showed numerous nucleated red corpuscles. Some of these were of large size, reaching  $20\ \mu$ , with irregular nuclei. There were also some cells containing fragments of brown pigment.

This case presented an excellent example of the characteristic fatty degeneration so common in fatal cases of pernicious anæmia. Its unfortunate termination simply repeats an occurrence with which all are but too well acquainted. When Hunter brought forward his views in regard to the nature and treatment of this disease many were full of high hopes that a satisfactory means of effecting recovery had been found. Results of a most satisfactory kind have been published by myself. It must be frankly, but sorrowfully, confessed that these hopes have proved vain, as further experience has failed to confirm the beneficial effects seen at first. Intestinal antiseptics, like all other remedies, only produce a transitory improvement, and we seem as far as ever from a reliable means of bringing about permanent recovery.

CASE 52. *Cardiac Dilatation from Fatty Degeneration.*—E. M., aged 55, housewife, was admitted to Ward 25 of the Royal Infirmary, on 2nd April 1894, complaining of breathlessness. Her father died early in life, of what affection the patient did not know. Her mother was alive, and, for the age of 75, enjoyed good health. There had been one brother, who died early in life, and one sister still alive and well. The patient had been the mother of eight children, of whom three died in infancy, and one boy at the age of 4. The other four children were strong and well. Her social conditions had been satisfactory. Her previous health had been good. Two years before her admission she began to suffer from some breathlessness, and she was at the same time heavy and sleepy. These symptoms passed off, and she gradually improved, remaining pretty well until shortly before admission, when she began again to feel breathless and weak.

On admission the patient was observed to be of small stature and of undue bulk, her height being 4 ft. 10 in. and her weight  $10\frac{1}{2}$  stone. Her face was deeply cyanosed, the lips being almost the colour of a ripe blackberry; while the ears and nostrils were of a dull purplish hue. The cheeks were livid, and marked by intensely injected arborescent veins.

The tongue, which, like all the mucous membrane of the mouth, was of a deep purplish hue, had a thin fur upon it. Many of the teeth were gone, and the gums were somewhat spongy. The liver was found to be rather enlarged, extending from the upper margin of the fourth rib downwards for  $6\frac{1}{2}$  in. in the mammary line. The spleen was also slightly enlarged, reaching the anterior axillary line. There was considerable distension of the viscera, but no fluid in the peritoneal sac. The pulse, which was 96 on admission, fluctuated during the week succeeding that date from 100 to 120. The vessel was somewhat rigid, and slightly tortuous. It was rather empty, but the pressure was fair. The pulsation was irregular and the beats unequal, being withal somewhat variable in type. There was seen to be a great distension of the jugular veins, which stood out like large knotted cords on the sides of the neck, with scarcely any perceptible movement. The præcordia showed no abnormal phenomena; in fact, no movement whatsoever could be detected. Palpation failed to detect any impulse. Percussion fixed the right border of the heart at  $2\frac{1}{2}$  in. and the left at  $4\frac{1}{2}$  in. from mid-sternum. On auscultation nothing could be heard except extreme feebleness of the heart sounds. At the base of the heart the first sound was almost inaudible, and the second sound was much louder in the pulmonary than in the aortic area. At the apex the first sound was dull in character and very feeble, and the second sound quite overshadowed it. The same was the case practically in the tricuspid area; but the first sound was a little more distinct.

The patient had a considerable amount of cough, attended by a fluid frothy sputum, slightly blood-stained. Physical examination of the chest only revealed the existence of numerous crepitations through both lungs, especially at their bases behind. The renal secretion was very scanty, never exceeding 14 oz. on any of the first three days after her admission. The subcutaneous textures showed considerable œdema of the dependent parts, especially of the ankles and feet. The patient was in a somewhat dull and listless mental condition, tending towards stupor.

It was obvious that in this case the myocardium had entirely given way, and that whatever might be the cause of the breakdown, there could be but one termination, unless speedy relief were afforded. The patient was therefore treated with Henry's solution, and a mixture was ordered, containing 10 minims of tincture of digitalis and 15 minims of tincture of squills, along with 15 grains of acetate of potash, in a tablespoonful of decoction of scopolarius, which was to be administered every four hours. As on the second and third days after admission there was no appearance of improvement, the patient, on the other hand, becoming more cyanotic and verging upon coma, it seemed to me that there was but one course open—general blood-letting. On the 4th April, therefore, Dr. Garbutt performed venesection on the left arm, and withdrew 10 oz. of blood. The effect of this was most marked, the patient becoming much more wide-awake. The cyanosis became less, the pulse gained in strength, and even the heart sounds were more distinct; while coincident with these symptoms of improvement there was a noteworthy increase in the renal secretion, which at once bounded up to 60 oz. The administration



of digitalis and squills, with acetate of potash and scoparius, was continued, and occasional doses of Henry's solution were administered ; and as there was still considerable crepitation over the base of the lungs, the back was dry-cupped. The cyanosis steadily disappeared, and all the symptoms ameliorated, the heart sounds in particular became very much more distinct ; but the digestive functions caused more trouble after a few days, probably on account of the digitalis. She was therefore treated thenceforward by means of tincture of strophanthus, 5 minims being administered every four hours. Although a few vicissitudes took place, her progress was eminently satisfactory, and it was discovered after she had regained a considerable degree of strength, that there were systolic and diastolic murmurs in the aortic area. By the 28th of April she was able to be sitting up, and she returned to her home on the 2nd of May.

She has since then reported herself from time to time, the last time on which she came to see me being 3rd November 1896, on which date she was free from all symptoms of circulatory disturbance, showed no trace of cyanosis or of venous engorgement anywhere, and the heart sounds were distinct and clear, except in the aortic area, where a systolic as well as a diastolic murmur could be detected.

It was certain that in this case we had to deal with a chronic myocardial alteration, but whether it was of the nature of a fibrous or a fatty change, could not be determined. Clearly the myocardial lesion was grafted upon an affection of the aortic cusps, almost certainly of sclerotic type ; possibly, therefore, a certain amount of interstitial myocarditis may have been present. From the general habit of body of the patient, it was certain, however, that fatty infiltration was present, and it seemed to me that the most reasonable explanation of the whole condition lay in an invasion of the muscle elements by fatty degeneration in consequence of the accumulation and infiltration.

### MYOCARDITIS.

Myocarditis presents itself in two distinct varieties, acute and chronic, which have, whether in respect of causation, structure, or effects, so little in common that they may almost be regarded as different diseases.

### ACUTE MYOCARDITIS.

In the pages of Morgagni and Senac there are clear indications that these observers were acquainted with such a



process, and Corvisart mentions it as an accompaniment of acute changes in the pericardium and endocardium. Laennec devoted an important section of his great work to the subject, and Bouillaud believed in its existence apart from acute lesions of the membranes. Hamernik described the microscopic appearances for the first time. Latham and Craigie threw much light upon the whole subject, while Stein carefully studied the various aspects of the disease in a luminous monograph. Since the date of his work, many observers, some of whom will be mentioned afterwards, have filled up the gaps in our knowledge.

A considerable amount of difficulty still surrounds the nature of this affection, inasmuch as some examples of the disease are mainly characterised by degenerative changes of the muscle cells with little or no structural alteration of the interstitial tissue, while in others the disease processes affect both muscular fibre and interstitial tissue in an almost equal ratio. Some instances might, therefore, almost be classed as an acute degenerative process.

ETIOLOGY.—The affection is always secondary to some morbid process elsewhere. It is most frequently found in connection with general or constitutional diseases. Scarlet fever, enteric fever, diphtheria, pyæmia, and rheumatism are the more frequent primary affections. Its occurrence, however, in the course of such affections is brought about in an indirect manner. There are two modes of origin. In one of these endocarditis or pericarditis has resulted from the primary disease, and by extension lights up the process in the adjacent muscular tissue. In the other, toxic influences are produced by emboli carried in the blood stream and deposited in the muscular tissues; this is more particularly the case in septic conditions. Traumatic influences are sometimes regarded as causes of acute myocarditis, but it is probable that in all such instances the disease has owned a double causation, an infective process having been present as well as the direct violence.

MORBID ANATOMY.—The structural alterations are sometimes general, involving the muscular substances indiscriminately; much more commonly, however, the changes are partial

and are confined to one region. If general in their distribution, they may affect the whole structure of the heart uniformly; if partial, there may be widely-scattered lesions of small size. When it results from pericarditis, it may be found widely distributed, but affecting only the superficial layers of the heart. During foetal life the right ventricle appears to be more frequently affected than the left, but after birth the converse takes place. The auricles are rarely affected.

The appearances presented by the disease yield considerable differences according to the stage which has been reached. In the earlier phases of the disease, the affected portions are of a deeper tint than in health, and the tissues are swollen and soft. Extravasation of blood is often seen in the affected part. At a later stage the tissue becomes paler in hue, often presenting a grayish appearance, and it becomes still softer than at first. Even with the naked eye, muscular fibres may be observed to be separated from each other sometimes by a serous infiltration containing blood corpuscles and proliferating cells. The change may go on to the formation of a true abscess, with so much destruction of muscular fibres as to form a cavity containing pus alone. This is more particularly the case when septic processes have produced the disease. Such abscesses have at times produced a connection between different cavities of the heart. It is stated by Gowers that, even after pus has been formed, caseation may occur, and the caseated mass may shrink and undergo calcification. On the other hand, the acute process appears in certain instances to subside leaving behind it a formation of increased fibrous tissue.

In acute myocarditis the essential alterations observed on microscopic examination vary with the intensity and duration of the disease. In an early stage the muscular fibres are larger and paler than in health, and the transverse striation is almost, if not quite, obliterated. Between the muscular fibres the interstitial tissue contains some proliferating cells mingled with red blood corpuscles and leucocytes. At a somewhat later stage the muscular fibres are granular and exhibit various stages of resolution into their component cells, separated by a collection of oil globules and masses of pigment. The interstitial tissue at this stage contains more leucocytes, but in some instances,

where conservative processes are in the ascendant, there is an increased amount of interstitial connective tissue. These changes are shown in Fig. 175. At a still later stage the process may advance to such a degree of disorganisation as to leave few traces of the normal structure, or, on the other hand, interstitial connective tissue may be so much increased as to form a fibroid degeneration of the heart.

It has been customary since the publication by Virchow of his views to separate acute myocarditis into

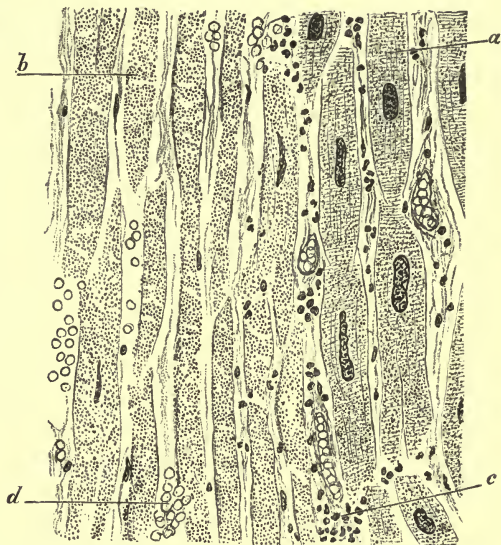


FIG. 175.—Section through wall of left ventricle in acute myocarditis,  $\times 250$ . *a*, Healthy muscle fibres; *b*, muscle fibres undergoing necrosis; *c*, accumulation of leucocytes; *d*, extravasation of red blood corpuscles.

parenchymatous and interstitial; but, as a matter of fact, both forms of the affection are associated together.

Certain secondary results of acute myocarditis are commonly found. In the more acute forms of the disease an abscess may lead, as above mentioned, to perforation, and fistulous communications may thus be established between different cavities. When the process is less acute there is a tendency to dilatation in consequence of weakening of the wall of the heart. From the diminished energy of the heart, blood-clots are apt to occur. Any, or all, of these effects may produce disturbance in consequence of involving the valves, chordæ, or capillary muscles, or by weakening the wall of the heart. It need hardly be added that in consequence of these disturbances remote effects are produced in other organs in consequence of retardation of the blood current and embolic



processes. In this way hyperæmia, catarrh, œdema, and infarction are common results.

SYMPTOMS.—The clinical features in acute myocarditis are certainly far from definite, and are characterised for the most part by lessened energy of the heart along with the general symptoms of acute disease.

The onset of the disease may be quite insidious, but is occasionally marked by characteristic features, occurring for the most part in the course of previous disease. The affection of the myocardium is sometimes announced by rigor followed by a rise of temperature; on the other hand, the temperature, previously elevated, may be still further increased without any shivering. The temperature in cases which have been recorded has been so different that it is impossible to attain to any definite conclusion with regard to it. According to Schrötter, the temperature as a rule is only slightly increased, but Wunderlich and Wagner have recorded one case in which the temperature, continuously elevated, sometimes reached 107°.

The digestive processes are usually interfered with, nausea and vomiting being frequent. Jaundice has often been observed. Painful subjective sensations connected with the heart have several times been noted. Dropsical appearances have been described. The pulse has usually been feeble, frequent, and irregular. The impulse of the heart has been imperceptible or extremely feeble. The cardiac dulness for the most part has been increased on account of dilatation. On auscultation the chief characteristics of the cardiac sounds have been weakness and irregularity. Sometimes there has been doubling of the second sound with accentuation of the pulmonary portion. Systolic murmurs are not infrequent, but Stein has observed that in cases with previous valvular disease the effect of acute myocarditis is to cause the disappearance of symptoms. Respiratory symptoms are common—breathlessness and cough, calling attention to disorders of the bronchial tubes, lung tissue, or pleural membrane. The commonest affections are bronchial catarrh, pulmonary œdema, and pleural effusions. Renal affections are very commonly linked with acute myocarditis. The urinary secretion is diminished, and contains



blood as well as albumin, while recently formed casts are also found. The nervous system is subject to many disturbances—headache, giddiness, and convulsions—or delirium, lethargy, and coma may be developed.

The course of the disease is, as a rule, rapid, and most cases appear to have terminated within a week. Oppolzer has indeed narrated an instance in which death occurred within a few hours of the onset of the affection. Demme, however, has recorded one in which the duration existed six weeks. Such examples are uncommon. Undoubtedly some local forms resulting from endocarditis or pericarditis undergo recovery, but it is probable that when the lesion is at all widely distributed an unfavourable termination must take place. Death usually occurs through cardiac failure, but it has sometimes been brought about by rupture of the wall of the heart. In some cases pulmonary or cerebral complications are an apparent cause of death.

DIAGNOSIS.—This is a question rather of theory than of practice, in consequence of the infrequency of the disease and the uncertainty of its symptoms. There can be no doubt of the extreme difficulty which surrounds the diagnosis. Many symptoms may be produced by other affections, while many instances of the disease are attended by extreme latency of clinical phenomena.

The rapid development of cardiac failure in the course of affections liable to be complicated by myocarditis, such as endocarditis or pericarditis among local diseases, and pyæmia or enteric fever among general diseases, might lead to the suspicion of acute myocarditis.

The differentiation of acute endocarditis and acute myocarditis is a matter beset by great difficulty, seeing that in both affections there may be an almost total absence of physical signs, and, even when physical examination reveals distinct morbid appearances, the evidence which these furnish may be conflicting. It may, for example, occur that in one of the general diseases liable to be followed by endocarditis or pericarditis, incompetence of the auriculo-ventricular valve ensues. In such a case it may be almost, if not quite, impossible to determine whether this complication is due to

muscular or to valvular affection. The diagnosis can therefore only be attained by carefully estimating the preponderance of evidence in favour of cardiac implication.

PROGNOSIS.—If it be possible to diagnose myocarditis, the question of prognosis must of necessity arise. It need hardly be said that in general or diffuse forms of myocarditis, arising from septic conditions, the prognosis is perfectly hopeless. It would be somewhat less grave in cases of local myocarditis if it were possible to determine the presence of such, while in the superficial form arising in consequence of pericarditis the outlook would be distinctly more favourable. Seeing that the diagnosis of such changes is absolutely uncertain, these remarks are purely speculative.

TREATMENT.—From the difficulty of recognising acute myocarditis, there can be no doubt that most instances have been treated for the primary affection which has been present rather than for the cardiac complication. Even in those cases in which it is suspected, if not determined, the treatment will necessarily be symptomatic. Absolute rest and appropriate diet must be enjoined. The use of cardiac stimulants will be demanded in consequence of the failing energy of the heart, and nerve tonics may well be combined with them.

Many local means have been advocated by different writers. The application of cold to the præcordia is sometimes thought to have some influence in curbing the disease. This may be so, but with the lowered vitality and usual uneasiness it is probable that heat would be more useful. Counter-irritation has also been recommended, but it is doubtful if any good can accrue from such local treatment.

### CHRONIC MYOCARDITIS.

Several distinct processes undoubtedly fall to be considered under this head, and a considerable amount of confusion is to be observed in the literature of the subject in consequence of the failure to discriminate between different, although related, lesions. The different terms which have been employed show how diverse are the opinions which have been expressed as

regards the morbid changes undergone by the muscular tissue. Cardiac sclerosis, cardiac cirrhosis, fibroid degeneration, sclerotic myocarditis—such are some of the names employed to designate forms of chronic myocarditis. It seems to me that in the light of our present knowledge the only philosophical way in which chronic myocarditis can be regarded is as including all chronic conditions in which there is a general or local increase of fibrous tissue. It is probable that all these conditions are connected with reaction phenomena.

Morgagni was the first writer to call attention to this affection, and since his time it has been referred to by most subsequent writers on cardiac affections, although it must be admitted that until comparatively recent times the disease was but little understood. Morgagni regarded the process as, in some respects, a degeneration. No real advance was made until quite recent times, and our modern knowledge has been somewhat rapidly developed. In connection with such a subject as this, presenting so many different aspects, it is by no means easy to arrange the work of the different authors who have thrown light upon the different lesions, and it would be impossible to mention them all. Only those, therefore, whose work is really helpful will be referred to.

The close connection existing between changes in the coronary arteries and fibroid lesions of the myocardium was observed by Gairdner a good many years ago, but he laid no stress upon the association. It was therefore left for Weigert, in his suggestive paper on tissue coagulation, to prove how intimately the lesions are related in the sense of cause and effect. He pointed out that when the circulation is gradually lessened by sclerosis of the arteries, wasting and destruction of the muscular fibres occurs, with consequent changes in the connective tissue. He showed, moreover, that in the case of sudden arrest of the circulation the muscular fibres and the connective tissue lose their nuclei as the result of acute necrosis, while yellow masses like coagulated fibrin make their appearance, around which, apparently from reaction, many round and spindle cells are to be found. Turner almost immediately afterwards dealt with the subject, and emphasised the close connection between the coronary circulation and the

myocardium, and suggested the possibility of the origin of local fibroid changes as a result of arterial obstruction or venous thrombosis, in consequence of injury from overstraining the walls of the ventricles.

Huber shortly afterwards discussed the question, and brought together the results of many observations; besides adding very largely to the facts of etiology as connected with the coronary circulation, he defined the relationship between the process and other conditions with which it has been confused. Letulle also entered upon the subject and laid great stress upon periarteritis in this connection as the starting-point of the change.

In this country the most valuable work on this subject has been done by Lindsay Steven, who has, in two important contributions, not only brought together a critical summary of the literature of the subject, but added materially to our knowledge of its pathology. Several facts brought to light by his industry will be discussed in the sequel.

Amongst authors who have thrown light upon different aspects of the subject may be mentioned Jenner, who has dealt with the results of long continued hyperæmia; Lancereaux, with important observations upon primary chronic myocarditis, as a result of toxic influence, and secondary, as a result of pericarditis or endocarditis; Fagge, with careful descriptions of the localised characteristics of fibroid changes; Thurnam, with observations on its association with cardiac aneurysm; Renaut, with his suggestive, but, as yet, unproven, observations on chronic segmentary myocarditis; and Ziegler, with his valuable contribution on myomalacia cordis from coronary changes.

ETIOLOGY.—It is an undoubted fact that families show a hereditary tendency towards chronic myocardial changes. After the most careful exclusion of every possible cause, there is sufficient evidence to show that a proclivity exists to such myocardial changes.

With regard to the influence of sex, the results of Lindsay Steven appear to agree pretty well in the main with those of other observers. In a series of 21 cases, 14 occurred in men and 7 in women. It is essentially a disease of advanced life,



and here again Lindsay Steven's facts may be appealed to. In 19 of his 21 cases, the age was ascertained. Of these, 3 were between forty and fifty years of age; 8 between fifty and sixty; and 8 between sixty and seventy. The age is stated by some authors as being without marked influence. This, however, is certainly wide of the mark, and there can be no doubt that occupations involving long continued severe muscular exertion are more likely to give rise to interstitial myocardial changes than any others.

The condition of the blood supply is of an importance which cannot be over-estimated. The blood itself may be modified by causes taking their origin within the body, or acting upon it from without, and the amount of blood circulated through the heart may be modified either by changes in the energy with which the blood is driven, or interferences with its local distribution.

Among factors modifying the condition of the blood arising within the body may be mentioned such affections as lithæmia, glycæmia, and all other faulty conditions of the blood arising from defective metabolic processes. Of those having their origin from without are some dependent upon the introduction of chemical substances, such as alcohol. It cannot be doubted that all agents which interfere with tissue change are liable to set up chronic lesions of the myocardium. In addition to these, some infective diseases are undoubtedly prone to give rise to chronic myocarditis, as for example enteric fever, as has been proved by Landouzy and Siredey. The most important of all the infective diseases in this regard is syphilis, first shown by Wilks, but its mode of action is somewhat indirect, since it acts both by the production of gummata, which form foci of irritation, and through obliterative endarteritis, which interferes with the nutrition of the part.

The supply of blood may be interfered with by causes which diminish the rapidity of its flow. Valvular diseases, especially those of the mitral valve, lead to venous stasis, in which the coronary circulation participates. Not only is the blood in such cases stagnant, but it is necessarily less pure, and, as a result, there is chronic induration of the walls of the heart, analogous to what is found in all other organs subject

to backward pressure. Similar conditions may be produced as secondary results of lesions elsewhere. In cardiac failure due to lung or kidney disease, analogous alterations are to be seen.

The influence exerted by the condition of the coronary arteries is the most important factor which has to be considered. All changes in the coronary arteries, whether affecting their walls or their lumen, lead to alterations in the myocardium. Endarteritis deformans, whether in the diffuse or nodose variety, with or without atheroma or calcification, is the most frequently observed vascular change. The presence of such chronic vascular changes is often associated with the existence of chronic renal disease, and indeed with the tendency to general fibrous increase throughout all the tissues of the body. Endarteritis obliterans is also sometimes found, and is attended by a development of gummata within the myocardium. The processes of thrombosis and embolism play an important part in the evolution of chronic interstitial myocarditis. Thrombosis may take place *in situ*; when the coronary arteries are seriously diseased by any form of endarteritis there is a great tendency to coagulation of the blood within the lumen of the vessel. Embolism may take place in consequence of valvular vegetations or coagulation of the blood within the cavities of the heart. In addition to all these different processes, chronic myocarditis undeniably results from extension. In cases of pericarditis with adhesions it may most particularly be seen, but in all cases of endocarditis and pericarditis there is more or less tendency to a spreading of the process into the adjacent layers of the myocardium.

MORBID ANATOMY.—Arising in consequence of so many different causes, it is little wonder that the appearances in chronic myocarditis are extremely varying. In almost all its forms it is a local change, but even in the most circumscribed form it produces alterations involving, in almost every case, a considerably larger area of the heart.

The structural alterations are practically confined to the ventricular portion of the heart. These morbid changes are far more common on the left than on the right side. Since the observations of Morgagni, it has been recognised that the

lesions are much more common near the apex and the inferior part of the ventricular septum than elsewhere. Chronic myocarditic changes are sometimes found, more especially affecting the right ventricle.

The heart in chronic myocarditis is almost invariably



FIG. 176.—Interstitial myocarditis from coronary sclerosis.

enlarged in consequence of dilatation; it may, or may not, be accompanied by hypertrophy. As a general rule, it is increased in weight as well as in bulk.

While the total volume of the heart is usually increased in chronic myocarditis, the affected portion of the walls is almost invariably found to be thinner than it should be. This is seen in Fig. 176. The diseased region is paler in colour than



the rest of the heart, having, as a rule, a grayish or yellowish tint. Occasionally there are small points deeply stained with blood in consequence of small seats of thrombosis.

The part involved generally gives a sensation of hardness when touched; but the variety particularly described by Ziegler under the name of myomalacia cordis yields to a

sensation of softness. According to Ziegler, it is simply an early stage of the sclerosis.

In this form of myocarditis the coronary arteries are almost always found to be the seat of alterations. The left coronary artery is that which is most frequently affected. The change may be at the orifice, which may be obstructed by atheroma, or the lumen of the vessel beyond



FIG. 177.—Section of wall of left ventricle in chronic myocarditis from coronary occlusion,  $\times 60$ . *a*, Sub-endocardial tissue; *b*, muscular fibres, largely altered; *c*, new fibrous tissue.

may be reduced by endarteritis. The changes, whether at the mouth or in the course of the artery, are often attended by deposits of lime salts; such are the arterial changes most commonly associated with the chronic sclerotic changes presenting hard and resistant masses. In the case of myomalacia, it is more common to find an embolus or a thrombus in one of the coronary vessels. Very frequently the aorta shows forms of degeneration, and it is not an uncommon experience to find the aortic cusps also diseased. Widespread sclerosis through-



out the arterial system, along with cirrhosis of the kidneys, and, in fact, an increase generally in the fibrous tissue throughout the body, may also be frequently determined. There are, moreover, in many cases, evidences of the secondary results of cardiac failure in hyperæmia and œdema throughout the body, while infarct is frequently to be found in some of the internal organs.

The microscopic appearances reveal atrophy of the muscular fibres, and increase of interstitial tissue. The muscle cells show different degrees of disintegration, varying from granular to fatty degeneration. Fibrous tissue, usually of a dense character, is found forming layers of varying thickness, embracing remains of what has evidently been muscular tissue, and spreading irregularly amongst

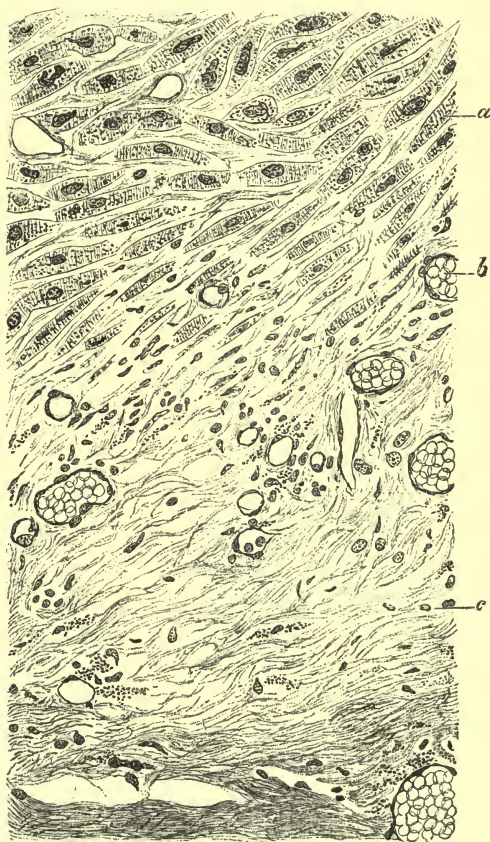


FIG. 178.—The same section under a higher power,  $\times 250$ . *a*, Muscular fibres, many undergoing destructive changes; *b*, engorged blood vessels; *c*, newly formed fibrous tissue.

muscular fibres undergoing degeneration. Such changes are represented in Fig. 177 and Fig. 178.

When any branch of considerable size is occluded—and this is usually caused by thrombosis within a sclerosed vessel, not by embolism, as Marie has most clearly shown in recent

times — infarction results. The appearances presented by the affected area differ according to the time which has elapsed after the stoppage has taken place. If seen shortly afterwards, the only change in appearance is concerned with colour, the part retaining its normal consistence, but being pale. When a longer interval has occurred, the part affected is soft and pale, the tint varying from pale brown to yellowish

white. Under the microscope the fibres are found to be disintegrated and altered. The striæ disappear, and they have a perfectly hyaline appearance. This is the *myomalacia cordis* of Ziegler. In the zone surrounding the affected area proliferation is found, and a reaction process of fibrosis is the result. Such changes are seen in Fig. 179.

**SYMPTOMS.** — Inasmuch as instances of chronic myocarditis constitute a somewhat irregular and rather indefinite group of affections, the clinical features which result are exceedingly variable, and it might be possible, as has indeed been done by some authors, to separate out several distinct types according to the main symptoms.

As the appearances presented by such different varieties pass gradually into one another, so as to form an almost continuous series, such a classification would not be so simple or satisfactory as might on the face of it at once appear.

In some instances, features of increased excitability and activity of the circulation—the *erethism* and *hyperkinesis* of authors fond of using learned terminology—make their

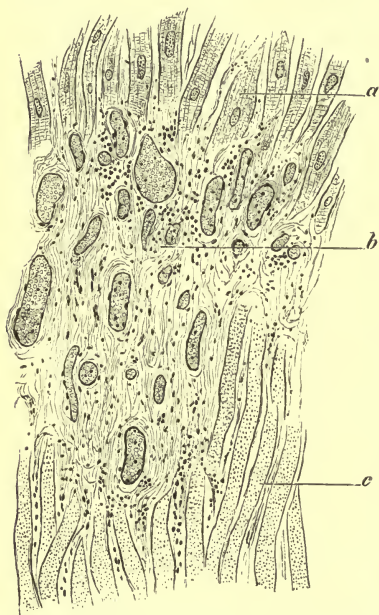


FIG. 179.—Section of left ventricle showing effects of infarct,  $\times 100$ . *a*, Healthy muscular fibres with distinct nuclei; *b*, young connective tissue with numerous vessels growing around the infarct; *c*, necrosed muscular fibres which are hyaline, and have lost their nuclei.

appearance. Here the main complaints are of palpitation of the heart, of throbbing of the arteries throughout the body, of surging noises in the ears, and even of flashes of light before the eyes. There may, on the other hand, be complaints of fluttering in the region of the heart, along with attacks of breathlessness, attended by giddiness and faintness. Both of these somewhat characteristic groups of symptoms are frequently attended by anginous attacks. There is no necessary disturbance of the functions of the alimentary system, unless cardiac failure be present, in which case it may be accompanied by its common manifestations as regards enlargement of the solid viscera, with catarrh of the mucous membranes, and the presence of some transudation into the peritoneum. The condition of the blood may, or may not, be normal. There is no necessary connection between it and the myocardial change. As above mentioned, painful sensations are often complained of, and these may present every feature of profound angina pectoris. Such cardiac pain may be accompanied by fluttering or palpitation of the heart, and there is not infrequently the alarming sensation of cardiac stoppage, along with the overwhelming sense of impending dissolution. The appearance of the patient presents nothing that is actually characteristic, unless during an anginous seizure. On close inspection a tortuosity of the temporal arteries may be determined, and there may be excessive pulsation in the jugular fossa, or of one or both of the carotid arteries, as well as sometimes venous pulsation in the neck. Inspection, as a rule, fails to reveal any abnormal phenomena connected with the præcordia. The radial arteries are commonly found to be somewhat degenerated, and are frequently hard and tortuous. The vessels are usually somewhat fuller than in health, and the pressure may be elevated. Infrequency of the pulse is relatively common, but occasionally the opposite condition of acceleration may be in evidence. It is, however, to be remembered that when cardiac failure is present, as is apt to be the case towards the termination of this affection, the pulse may show exactly opposite conditions, that is to say, it may be somewhat empty and of low pressure, while the pulsation is marked by irregularity and inequality. The apex beat is generally displaced somewhat outwards



to the left, as well as downwards, since either hypertrophy or dilatation, or both, may be present. The area of cardiac dulness is likewise usually increased. The cardiac sounds are almost always affected. Perhaps the most frequent manifestation of disturbance is an accentuation of the second sound in the aortic area, along with doubling. In the mitral area the first sound is usually lower in pitch than in health, and somewhat muffled; but a systolic murmur is extremely common, in consequence of relative incompetence of the mitral cusps as a result of cardiac dilatation. A systolic murmur in the tricuspid area may often also be determined.

Dyspnœa, whether shown in the form of breathlessness on exertion, or as a recurrent asthma, is often developed, and it not infrequently happens that Cheyne-Stokes' respiration makes its appearance whenever the patient falls asleep. Cough frequently calls attention to some changes in the condition of the lungs, such as bronchial catarrh or pulmonary cedema, which may be determined by examination; hydrothorax is also often present.

The functions of the kidney are usually disturbed, the secretion being of diminished quantity and high specific gravity; containing besides, in many cases, albumin.

In addition to the painful subjective sensations, other nervous disturbances are found, more particularly sleeplessness, restlessness, loss of memory, and other expressions of cerebral trouble. The course of the affection differs much in individual instances. It may often pursue a somewhat lengthy course, and end in a gradual cardiac failure, attended by many of the symptoms above mentioned. It may, on the other hand, terminate in sudden death.

DIAGNOSIS.—In all these symptoms there is really nothing that is characteristic, and this indefiniteness of the clinical features constitutes the chief difficulty in the determination of the nature of the affection. It is, for instance, by no means an easy task to differentiate between chronic myocarditis and fatty degeneration. There can be no doubt that in the latter condition it is not so common to find evidence of arteriosclerosis. Yet, it must be remembered, fatty degeneration often occurs along with the cardiac hypertrophy attendant



upon these two conditions. It will, notwithstanding, be admitted that when there is arterial degeneration with a fairly vigorous cardiac systole, and an accentuation of the aortic second sound, associated with some cardiac pain, the probability is strongly in favour of the condition being one of chronic myocarditis rather than of fatty degeneration. The chief difficulty comes in when, as the final result of chronic myocarditis, cardiac failure ensues. The differentiation of certain forms of valvular disease and fibroid myocarditis is also by no means easily effected. Taking, as an example, the physical signs of mitral incompetence, it may be a matter of the utmost difficulty to arrive at a conclusion whether this is produced by some lesion primarily of valvular origin, or is a relative incompetence produced by dilatation of the orifice or ventricle. The history of the case, however, comes to the aid of the observer in such an instance, and when there is no distinct history of a valid cause of valvular disease; when there is, further, no evidence of there being any obstruction, and when some of the symptoms of fibroid myocarditis, as above described, have been developed, it may be possible to conclude that the lesion is due to a myocardial affection.

PROGNOSIS.—When a diagnosis of chronic myocarditis has been achieved, the prospects of the patient will next require consideration. Probably the history of the affection constitutes a more useful guide in attempting to formulate a prognosis than the mere condition which may be present. If the condition has evidently only been developed after the lapse of a considerable time, and if the cardiac impulse and first sound are satisfactory, the outlook may not be especially gloomy. The effect of muscular exertion should always be ascertained, and if the result of walking upstairs, or some other gentle exercise, be to cause any considerable perturbation of the cardiac action, it means that the myocardium is inadequate. Attacks of angina pectoris and syncopal seizures are of evil omen, and when such are present they necessarily render the outlook more gloomy.

TREATMENT.—The treatment of chronic fibroid degeneration must be, to a considerable extent, conducted on the same lines as our experience shows to be useful in cardiac failure.

The careful regulation of all the habits with regard to rest and exercise, food and drink, must also be enjoined here. Every means by which the metabolic processes throughout the body can be stimulated must be employed. The use of abundant diluents will be found of great utility, and general massage may also be employed with the greatest benefit. When dealing with cases in which it appears to be satisfactorily determined that the myocardial change under discussion is in progress, but has not gone too far, the employment of baths, such as those of Nauheim, may be safely recommended, and along with them resistance exercises may be advised. Amongst drugs at such a period, beyond the use of gentle salines, there is but one which promises any good effects—that is, iodide of potassium. When this is continued over a long period in small doses, it undeniably is of considerable benefit. The various symptoms are lessened, and the heart in particular certainly gains energy. It is more especially useful when there are any anginous threats. It need hardly be said that in such cases manifesting the symptoms of cardiac failure, the use of the cardiac tonics, and, in short, all the methods applicable to failure of compensation, must be had recourse to.

CASE 53. *Chronic Myocarditis from Coronary Obstruction*.—D. S., a tinsmith, æt. 36, complaining of cough and breathlessness, was admitted to Ward 22 of the Royal Infirmary under my care on 12th March 1893.

The patient seemed to have no hereditary tendencies to disease, and, until a short time before admission, his surroundings were good in all respects. His previous health was excellent, and, in particular, although he had complained of vague pains in the joints and muscles, he had never suffered from any definite rheumatic attack. For a few months before coming to the hospital he had been ailing, but, as his wife was confined to bed on account of a severe illness, he had struggled on until, a fortnight before his admission, he had been compelled to take to his bed. In addition to the cough and dyspnoea, the patient complained of weakness and sleeplessness.

The appetite was very poor, and the patient had frequently vomited undigested food. The bowels were constipated. The liver was slightly enlarged, extending from the fourth rib to an inch below the costal margin in the mammary line. There was no ascites. The patient was distinctly anæmic, and so pasty looking as to suggest renal disease. There was no morbid appearance connected with the spleen or glands. Dyspnoea

was constant, but with paroxysmal exaggeration, especially at night. Some præcordial pain and considerable palpitation were complained of.

The neck and chest yielded no abnormal symptoms, except that the cardiac impulse was extremely weak. The radial arteries were somewhat hard, but the vessels were unfilled, and the pressure was moderate. The pulse was extremely frequent, varying from 130 to 150; it was perfectly regular, and the wave small. Cardiac dulness extended from  $2\frac{1}{2}$  in. to the right to  $4\frac{1}{4}$  in. to the left of the mid-sternal line. The heart sounds were extremely faint. A soft systolic murmur was heard over the præcordia, but it was so feeble that it was difficult to follow its distribution with accuracy. There could be no doubt that it was loudest at the lower end of the sternum, but there was a probability that there were two other points of maximum intensity, one towards the apex of the heart, the other over the manubrium.

Absolute dulness was present at the bases of both lungs, along with almost total suppression of the breath and voice sounds, on account of double hydrothorax, and many crepitations were to be heard above the level of the fluid on either side. The urine was scanty, varying from 18 to 24 oz. per diem. It contained no albumin or tube-casts. Considerable œdema of the subcutaneous tissues was present in the lower limbs. The patient complained much of sleeplessness and restlessness.

The diagnosis arrived at was cardiac failure, with mitral and tricuspid incompetence, in consequence of arterial degeneration, probably attended by some changes in the aortic cusps from chronic atheromatous processes.

The patient was treated by means of cardiac tonics: strophanthus was administered, as well as iron and strychnine, but his condition did not improve. Stimulants were also freely exhibited—alcohol, ether, and ammonia being given at short intervals. Although the patient slept better than before admission, the dyspnoea became worse, and on Sunday, 19th March, the breathlessness was so distressing that the resident physician, Dr. Donald Macaulay, found it necessary to remove some fluid from the right pleural sac by aspiration. Some temporary relief was obtained, but in a very short time pronounced œdema of both lungs followed, with a copious expectoration of frothy sanguineous fluid. The stimulation was increased, and gentle counter-irritation employed, but the patient's condition became rapidly worse, and he died suddenly on the following day.

The following is a summary of the chief facts observed at the post-mortem examination, which was performed by Dr. Muir on the 21st March.

The body was well nourished, and there was marked dropsy of the lower limbs.

*Heart.*—The pericardium was normal. The heart was considerably enlarged, and weighed 15 oz. The right side was distended with dark clot; the left ventricle, on the contrary, was rather collapsed, and its wall could be felt to be thin, especially along the anterior aspect, where it also had a peculiar stiff consistence. The aortic valve was very slightly incompetent, not sufficiently so to be of any importance. Diameters of



orifices : aortic, .9 in. ; pulmonary, 1 in. ; mitral, 1.2 in. ; tricuspid, 1.8 in. The aortic cusps were slightly thickened at their margins, otherwise the valves were normal.

The left ventricle was considerably dilated, measuring  $4\frac{1}{4}$  in. in length, and its width was increased even more proportionately. Its wall was much thinned, especially along the anterior division of the coronary artery, which ran further from the septum than usual. The wall here measured only  $\frac{1}{4}$  in. in thickness. This part was distinctly tough, and apparently fibrous, as was definitely found to be the case on microscopic examination. The muscoli papillares were exceedingly atrophied, and in part fibrous at their extremities : the columnæ carneæ were much thinned, some of them being almost like paper. The myocardium was rather pale and soft in those parts where there was no fibrous change. There were some ante-mortem thrombi adherent to the endocardium in the apex of the ventricle. The naked-eye appearance of the left ventricle may be seen in the illustration (Fig. 176). Part of the ventricle was removed for microscopic examination before it was photographed, and the thinnest portion is therefore not seen. It will, however, be observed that the walls are very slender, and that the papillary muscles and columnæ carneæ are remarkably small.

The right ventricle was of normal size, but its wall was much hypertrophied, its thickness being  $\frac{1}{4}$  in. at places, and the columnæ carneæ were thick and powerful, contrasting markedly with those of the left ventricle. The auricles were almost normal, the right being slightly dilated.

The aorta at its commencement showed extensive atheroma, chiefly in the form of soft cushion-like patches, with very little calcareous change. The orifice of the left coronary artery was in the centre of one of these patches, and was so much narrowed that it only admitted the entrance of a fine bristle. Immediately beyond, the coronary artery was of normal width, and in its course nothing abnormal was visible. The right coronary artery was not affected. On injecting the right coronary artery with water, a very small quantity passed into the left, showing the existence of a slight anastomosis.

There was hydrothorax on both sides, the left pleura containing 12 oz., and the right 20 oz. of serous fluid. The lower lobe of the right lung showed commencing hypostatic pneumonia. In both lungs there was congestion, with some œdema posteriorly. Only slight emphysema was present. The other organs showed chronic venous congestion. For the microscopic examination my best thanks are due to my colleague, Dr. Muir. The case was so interesting that we deemed it our duty to report it in the *Edinburgh Hospital Reports*.

Portions of the left ventricle, taken chiefly from the anterior wall, were examined both in the fresh condition and after fixing in corrosive sublimate and spirit. The changes found may be described as twofold, viz. extensive atrophic and degenerative changes in the muscle fibres, and marked interstitial growth of fibrous tissue in certain parts of the heart wall. The fibrous change was most marked towards the inner part of the



wall, and at many places a distinct though not uniform layer of fibrous tissue was present a short distance from the endocardium. In this fibrous tissue, which was for the most part of very dense character, there were visible merely the pigmented remains of muscle fibres and a few blood vessels. Fibrous tissue was seen at places spreading inwards irregularly and diffusely between the degenerated muscle fibres to the endocardium, and also running outwards along the intermuscular planes as more distinct bands which also invaded the muscle bundles. This fibrous overgrowth reached its maximum at the anterior branch of the coronary artery, where the dense layer came right up to the surface of the heart, so that at this part the wall was fibrous in its whole thickness, with the exception of a layer of degenerated muscle on the inner aspect. Here also the wall was thinnest.

The most marked degenerative changes were found in the innermost part of the wall, *i.e.* internal to the dense fibrous layer. In fresh sections the muscle fibres there showed extensive granular degeneration and disintegration; some of this was fatty in nature, and in places amongst the fibrous tissue the muscle fibres appeared simply as rows of granules or small globules. The degeneration was also marked in the papillary muscles, which also showed fibrous thickening here and there on their surface. These parts, in sections cut in paraffin, showed shrivelled muscle fibres, which appeared as if they had lost their sarcous substance, only a sort of envelope remaining, and which, with the rubin and orange stain, were coloured only a faint pink instead of the usual deep orange. Immediately superficial to the fibrous planes, muscle fibres could be seen of nearly normal appearance, but, in places, of extreme tenuity, with thin fibrous bands spreading between them. But an interesting fact is, that in some places it was quite evident that an extreme atrophy of the fibres preceded and was not caused by the fibrous overgrowth. At no place was there any collection of necrosed fibres, such as are seen in infarcts in the heart wall. Beneath the epicardium the muscle appeared fairly healthy, some of the fibres, however, appearing rather hypertrophied and their nuclei large and somewhat vacuolated. These facts are well shown in Fig. 177 and Fig. 178, which are drawn from sections of the heart now described.

The condition of the arteries was the following. The large branch on the anterior wall and its branches beyond, showing very slight periarteritic and endarteritic thickening at some places, were practically normal. At no place was there any distinct encroachment of their lumen, or any thrombosis. In the dense fibrous tissue, as one would expect, some of the capillaries and small arteries were lying compressed and closed, but even there many of the arterioles had quite normal walls, with wide lumen. In short, there was no evidence of there being any primary lesion of the branches of the coronary arteries of importance in relation to the other changes.

The most important points of clinical interest to be drawn from the study of this case are connected with the physical signs. There is one subjective symptom, however, which

must not be passed over without comment. The presence of præcordial pain has been noted in most of the cases of this nature which have been placed on record. It was, unfortunately, impossible to trace out the exact distribution of the pain in the present instance, and it is therefore only possible to make simple mention of the symptom. It was not of a severe character. As regards the physical signs which were observed, the first point worthy of notice is the fact that the pulse was throughout absolutely regular. In cases of cardiac failure, irregularity of the heart's action is extremely common, and it is a matter of interest to observe that in this patient there was no approach to arrhythmia. The only remaining aspect of the case requiring remark from the clinical point of view is the entire absence during life of evidence of aortic regurgitation. There was nothing resembling the water-hammer pulse of aortic incompetence, and no diastolic murmur could be heard. The reason for the absence of both phenomena is probably to be sought in the pronounced condition of cardiac failure.

### CARDIAC DILATATION.

Referred to by the older anatomists, dilatation of the heart was more particularly noticed by Harvey and Willis. Mayow described dilatation of the left auricle and right ventricle as a result of mitral obstruction, and Vieussens mentioned an instance of dilatation of the left ventricle resulting from aortic regurgitation. The method of production was carefully analysed by Senac, and the effects produced on the circulation were detailed by Morgagni. All the great writers upon diseases of the heart, from his time downwards, have dealt fully with the condition.

The first recognition of the clinical features of cardiac dilatation are really due to Corvisart, who observed the increased area of pulsation. The means of recognition by auscultation was carefully studied by Laennec. Bertin, in addition to extending previous observations upon the symptoms of dilatation, distinguished between different varieties,

and his classification has been very generally accepted, even to the present time.

ETIOLOGY.—The causes of dilatation of the heart constitute an extremely large group of factors, many of which have already been seen to be operative in the production of the atrophic and degenerative processes already passed in review.

The *predisposing causes* may be arranged under three classes, according as they are personal factors affecting the individual, general factors influencing the system, or special factors connected with the heart itself.

There can be no doubt of the influence of heredity as a personal factor. There is not merely an inherited tendency towards certain tissue changes, whereby atrophic and degenerative processes are liable to occur; but there is, in addition, a liability in certain families towards diseases apt to induce cardiac changes. The result of these two inherited tendencies is to render certain individuals more than usually liable to suffer from dilatation. In consequence of the greater exposure to physical stress, the male sex suffers more frequently from cardiac dilatation than the female. It cannot be that there is any greater liability primarily, yet under the social conditions which obtain, men are much more commonly affected by this lesion than women. Dilatation is more common at two epochs of life than during the remainder. During the period of adolescence, when all the different functions are undergoing adaptation, dilatation is more easily induced than during the prime of life. Again, in elderly people, in consequence of the incidence of atrophic and degenerative processes, dilatation again becomes more common. Occupation plays also an important part. Those who are engaged in work which entails considerable physical stress are much more liable to cardiac dilatation than those whose occupation does not expose them in this way.

The febrile condition amongst general factors, when severe or prolonged, has a powerful influence in the production of dilatation. In many forms of pyrexia, it must be allowed that the determining influence is probably toxic in its nature, and that, in many cases in which a fever produces dilatation, the etiological factor belongs to the group to be mentioned

immediately. In the present state of our knowledge, however, we must grant the possibility of pyrexia taking its origin in chemical changes, and nervous influences, which may have no connection with any micro-organisms. Toxic conditions are extremely important. Such may be microbic in their nature, consisting in poisonous alkaloids, albumins, and ferments, and there are also the various chemical poisons, of which alcohol may be taken as the type. All conditions in which the nutritive value of the blood is lowered are important in this connection. Poisonous agents may arise within the system. Probably the best example of such a process is to be found in the effects of renal disease. In acute and subacute nephritis, dilatation of the heart is not uncommon, usually with, but often without, any obvious hypertrophy. It has always seemed to me extremely probable that the occurrence of this condition is due to the action of retained waste products upon the heart muscle. Anæmia of every kind, leucocythæmia, adenia, and all other conditions in which there is transitory or permanent reduction in the amount of hæmoglobin, exercise an important influence in the etiology of dilatation. All cachetic conditions, by their interference with the nutrition of the blood, may give rise to the changes now under consideration. Over and above all these conditions, it must be admitted that a state of general laxity of the tissues may have its origin in defective hygiene from errors as regards food, drink, and exercise, so that when any exciting cause of dilatation supervenes, the cardiac muscle is apt to yield.

There are some important causes belonging to the group of special factors connected with the mechanism of the heart, whereby there is some interference either with blood supply or blood pressure. Some of the factors to be considered in this place might almost be relegated to the position of exciting causes, yet viewed carefully there can be no doubt that they are to be regarded as predisposing causes. The powerful influence of interference with the coronary arteries has been sufficiently insisted upon in connection with the lesions previously described, and nothing need be added in this place, save the statement that such interferences are quite as potent in the production of dilatation as of any of the other processes



already considered. It must be borne in mind that endocarditis, through implication of the myocardium by extension, may directly give rise to dilatation from myocarditis. Some affections of the pericardium demand brief consideration. If pericarditis involves the superficial layers of the myocardium, there may be a myocarditis giving rise to dilatation. In addition to this, there can be no doubt from the statistics of Kennedy and Hayden that pericardial adhesions are not infrequently causes of dilatation, the mechanism of which will be discussed immediately.

All these various predisposing causes give rise to dilatation by producing the conditions which have been previously studied, that is to say, weakness, atrophy, degeneration, and myocarditis.

Lesions of the orifices and valves of the heart constitute most powerful agents by furnishing such disordered mechanism as allows the disturbance of blood supply and blood pressure, which act as exciting causes. The relative importance of the different kinds of lesions will be discussed below.

The *exciting causes* are essentially connected with changes in the amount and pressure of the blood. The most influential condition is over-distension, but this must be accepted with some qualification, since over-distension is a somewhat relative term, and its significance depends upon the proportions existing between the amount of distension and the resistance of the walls. There may, therefore, be the interesting result that with a relatively low blood pressure, as for instance in the later stages of a fever, there may be considerable dilatation; the explanation of this is, that the weakness of the cardiac walls does not allow them to expel all the contents of the chambers, so that there is a remainder, which in relation to the energy of the heart may be sufficient to produce a condition of over-distension.

Acute exciting causes are usually connected with two conditions—a sudden physical stress, or a rapid heart failure. The former condition may be produced in almost any heart, no matter how great its energy, if the over-stress is in such a degree as to overcome the cardiac resistance. As examples of such causation may be cited severe muscular efforts of every

kind, such as over-training, long marches, carrying heavy weights, and many other severe muscular efforts. The manner in which such influences act in producing their effects is doubtless by increasing the general blood pressure. It is rather difficult at first sight to see how such a rise of blood pressure should be produced by exertion, seeing that a muscle in a state of activity has always an increased blood flow through it, and it would therefore be naturally expected that although the heart must of necessity act more frequently, and more powerfully to keep up the blood supply under such circumstances, the pressure would not unavoidably increase. There is one consideration which must not be overlooked—while the effect of stimulating a muscle in experimental physiology is to accelerate the blood flow through it, the effect of long-continued muscular action must be to form a large number of waste products, and it will necessarily follow that the blood may have greater difficulty in passing through the tissues after it has become loaded with such substances. In this way, the pressure will probably be raised. The mere muscular exertion, moreover, involved in long-continued efforts, is to produce increased intra-thoracic pressure, and this, added to the greater impurity of the blood, will interfere with the functions of the right side of the heart.

Such seems to my mind the rational explanation of the undeniable facts. The determining factor in such conditions of stress leading to strain is, without question, a residuum of blood which the heart is unable to expel, and which, therefore, leads to over-distension. The latter condition is almost invariably brought about, either by pyrexia, or by direct loss of blood. The mechanism through which a heart enfeebled by pyrexia becomes dilated, is probably by means of the retention of a considerable quantity of residual blood. This may in itself not be very much, yet, in relation to the cardiac energy, it may be sufficient to produce so much distension as to lead to dilatation. It undoubtedly appears somewhat strange that a severe loss of blood may give rise to dilatation, seeing that the first effect of such loss must be to diminish the total quantity of blood in circulation. A few moments' reflection, however, will recall the fact that after any drain of blood from the

system, there is an extremely rapid withdrawal of fluid from the tissues, so that the quantity of blood is augmented. As is well known, the blood is hydræmic, and there is probably much less affinity between it and the tissues, so that there is an interference with its passage through them. This is the probable explanation of the fact that in many cases of symptomatic anæmia, the blood pressure is rather above than below the normal. In such cases, there can be no doubt also that the nutrition of the heart must suffer from the diminished nutritive value of the blood, and if there be, therefore, increased resistance in front and lessened energy behind, an accumulation in the chambers of the heart is unavoidable.

The chronic exciting factors in producing dilatation may be connected with the heart itself, or with any part of the circulatory mechanism. All valvular lesions have a tendency to produce dilatation by causing accumulation within the chambers of the heart,—the retro-dilatation of Adams and Forget,—but they do so in very varying degrees. This is not the place to discuss the relative influence of obstruction and regurgitation—it will be considered in dealing with the morbid anatomy, yet it may be stated that obstruction is a less potent factor than regurgitation, no doubt because it has less tendency to produce residual accumulation. Pericardial adhesions are undoubtedly a cause of dilatation. The reason for this is obviously that such adhesions may interfere with the contraction of the heart, and with the complete emptying of the chambers, so that some residual blood is left, leading to distension. Pulmonary disturbances interfere with the right side of the heart, giving rise to dilatation of the right ventricle, and frequently also of the right auricle. The most common disease belonging to this group is emphysema, which undoubtedly acts by destroying such a large proportion of the pulmonary capillaries; it thus dams the blood back in the right chambers.

All affections of the systemic vessels, when affecting a wide area, may produce similar effects upon the left chambers. It is purely a matter of relation between peripheral resistance and central energy. If with a considerable increase of peripheral resistance there should be any enfeeblement of the

heart, in consequence of atrophic or degenerative processes, dilatation may be the result.

MORBID ANATOMY.—In considering the morbid appearances found in cases of cardiac dilatation, it is necessary to bestow attention upon several points—the size of the heart, both in whole and in part; the weight of the organ; the thickness of its walls; and the consistence of its texture.

It is rare to find any considerable dilatation of one chamber by itself. Most commonly every part of the heart is involved, but the part which is most subject to the influences at work suffers most markedly. It has been shown repeatedly that an obstruction is more likely to produce hypertrophy without dilatation, while regurgitation is more commonly followed by dilatation of the chamber into which the regurgitation takes place, attended by more or less hypertrophy.

In cases of aortic obstruction there is comparatively little tendency towards dilatation, and the chief change is more or less hypertrophy of the left ventricle, with but little alteration in any other chamber. Aortic incompetence almost invariably produces great dilatation of the left ventricle, accompanied by a high degree of hypertrophy. In mitral obstruction there is comparatively little dilatation of the left auricle, hypertrophy in a more or less pure form being present; while in mitral incompetence there is a very high degree of dilatation of the auricle. The fact must not be overlooked that in mitral incompetence the left ventricle is very commonly—indeed almost invariably—dilated as well as hypertrophied, the reason for which appears to be that there is in this affection such a large residuum of blood.

The right ventricle is very frequently involved as a consequence of affections of the left side. Aortic disease does not, as a rule, produce much influence upon the right chambers, unless the left ventricle has yielded, in which case the effects are practically those of mitral disease—aortic lesions with mitral symptoms, as Broadbent terms them. Mitral obstruction and incompetence, more particularly the latter, exercise the most powerful influence upon the right ventricle, by interfering with the onward passage of the blood in the lungs, and in these affections there is almost invariably some dilatation of



the right chambers. In the lung affections which are likely to give rise to dilatation, there may be great enlargement of the right ventricle, usually attended by more or less hyper-



FIG. 180.—Great dilatation of the right ventricle, with some hypertrophy, from a case of emphysema.

trophy, and it is common to find that both in affections of the left side of the heart, and of the lungs, the auricle, as well as the ventricle on the right side, are dilated.

The change in size is usually accompanied by some alteration in form, but this is more especially seen when the right side of the heart is particularly affected, seeing that left-

sided dilatation is commonly followed by that of the right. In aortic incompetence, for example, it is a very common thing to see hearts enormously enlarged from dilatation of every chamber, while the relative proportion of each part of the heart is but little modified. In mitral incompetence, more particularly, the heart does undergo some alteration in form, since the left auricle and the entire right side of the heart are more likely to be modified than the left ventricle. When dilatation results from any pulmonary condition, the right side of the heart may be almost solely affected, and it is sometimes so extremely large, as is seen in the illustration (Fig. 180), as to entirely overshadow the left. In such a case the right ventricle often forms the apex of the heart, and constitutes the vast mass of the organ. The size of the chambers has already been given in the anatomical section, but it may be stated here that the average measurements of the ventricular cavities taken from the apex of the cavity up to the base of the nearest sigmoid cusp is, according to Hamilton, on an average, for the left ventricle,  $3\frac{1}{4}$  in. in men, and 3 in. in women; while those of the right ventricle average  $3\frac{3}{8}$  in. in men, and  $3\frac{1}{16}$  in. in women. These measurements are often greatly exceeded in dilatation.

The weight of the heart in simple dilatation is not increased. This, however, is to be regarded as a theoretical speculation more than a scientific fact, since some hypertrophy is almost invariably an accompaniment of dilatation.

The auriculo-ventricular orifices undergo changes in dilatation of the cavities, and their dimensions are considerably increased. As a consequence, the cusps are rendered incompetent. This is not only the result of an enlargement of the orifices, but it occurs in consequence of the dilatation of the cavities by means of which the attachment of the papillary muscles to the walls is removed to a greater distance than in health. Sometimes this is to some extent obviated by degenerative processes in the papillary muscles, whereby they do not contract, and, therefore, allow greater possibility of the cusps meeting. Both fatty and fibrous changes in the papillary muscles may effect this change. If there be much dilatation of the auricles, there is usually considerable distension and

enlargement both of the auricular appendices and of the great venous channels.

In simple dilatation the walls ought necessarily to be somewhat thinner than in health. This, however, may very safely be stated never to occur. As has already been shown, the thickness of the left ventricle is, according to Hamilton, on an average about  $\frac{1}{4}$  in. at the apex and  $\frac{1}{2}$  in. at its thickest part near the base, both in men and in women, while the right ventricle is on an average  $\frac{1}{8}$  in. thick in both sexes, but parts of the right ventricle were found by this observer to cover sometimes  $\frac{1}{4}$  of an inch, or as little as  $\frac{1}{16}$ .

The conditions commonly associated with dilatation require but little comment. Many of them are etiologically linked with the dilatation; others, however, are its results, but as these latter are essentially produced by venous stasis, there is no necessity for following them out in this place.

The consistence of the walls of the heart in dilatation, when accompanied only by a slight degree of hypertrophy, and not attended by any considerable degenerative process, is found to be somewhat flaccid, but they are not markedly friable, or very easily torn. In such instances, the appearance of the muscular tissue may be perfectly normal, but it is very often somewhat pale, and occasionally mottled. On microscopic examination, the muscular fibres are often found to present indistinctness of striation, and granules or globules may be found in the cells. There is very often an increased amount of fibrous tissue between the muscle fibres.

The results of dilatation, produced by diminution of the driving power of the heart, are far reaching. There is a larger quantity of blood to be moved, while there is less energy to overcome its inertia, and in consequence, unless there be considerable hypertrophy as well as dilatation, the amount of blood driven onwards is less; as more blood, besides, is retained within the dilated cavities, the amount which is allowed to enter is diminished. Thus there is obstruction to the onward flow of the blood, with an excessive amount of blood in the venous, and a deficiency in the arterial, vessels. There are in consequence the various pathological effects of venous stasis. If the left ventricle be primarily affected, the left auricle is

first involved. The pulmonary veins also become dilated, and usually sclerosed; the pulmonary capillaries develop varicose appearances; the serum of the blood transudes into the pulmonary tissue, and hæmorrhages take place; while at the same time there is an increase in the connective tissue going on to the production of brown induration.

The effects are also carried towards the right side of the heart, and the ventricle becomes in turn dilated. The auricle follows, and then the coronary and systemic circulations become involved. In consequence of backward pressure upon the coronary sinus, there is an increase in the amount of the fibrous tissue of the heart, in short, one of the forms of interstitial myocarditis is the result. This, however, is somewhat later in its development than are the other effects of backward pressure, as the manner of opening of the coronary veins has almost the effect of a valve. The backward pressure tells in the first instance upon all the great veins, and the jugular veins of the neck are sometimes enormously distended. The liver suffers greatly in consequence of its close connection with the inferior vena cava. There is enlargement of the whole organ more or less uniformly in consequence of backward pressure upon the radicles of the hepatic vein, attended by the development of fibrous tissue around the vessels and between the lobules, and by fatty degeneration of the cells. Thus is produced the well-known nutmeg liver. There can be no doubt that sometimes the liver becomes ultimately reduced in size in consequence of fibrosis and atrophy. From the interference with the passage of blood through the capillaries of the liver, the whole portal system becomes implicated, and the spleen, stomach, and intestines suffer from venous stasis. The spleen becomes enlarged and indurated from increase of fibrous tissue, but it is to be observed that it never attains the same dimensions in cardiac disease as it does in alcoholic cirrhosis of the liver—a fact which Parkes Weber has lately sought to explain by the influence of some toxic substance in the latter case. The whole viscera become disorganised by catarrhal processes affecting their mucous membrane, and by hyperæmia of the peritoneal covering, leading to effusion into the abdominal cavity. The kidneys are enlarged and dark from distension.



In an early condition they are more friable than in health, and the capsule can be taken off readily, but later on there is induration from increase of fibrous tissue, so that the kidney finally may come to be smaller and tougher than in health, while the capsule will not easily strip off. Even the brain is subject to alterations, but these are not so distinct as in other organs, probably as the result of gravitation. There is, nevertheless, some distension of the veins, along with a wasting of the superficial layers of the cortex, and diminution of the cerebral consistence. Œdema of the subcutaneous tissues takes place, and it is more especially found in the dependent parts of the body.

SYMPTOMS.—It is never an easy matter to determine how many of the clinical features present in any given case are produced by dilatation, since it is so often accompanied by hypertrophy, and both conditions are so frequently associated with valvular lesions. It is further to be remarked that dilatation is often an attendant upon some of the atrophic or degenerative processes.

There is loss of strength and energy, and any call for increased exertion is apt to be followed by feelings of faintness, or by actual syncope. The digestive functions are almost always impaired, and various symptoms of gastro-enteric disorder are present, in consequence of the catarrhal processes in the mucous membranes. The area of hepatic dulness is almost invariably increased, and some ascites is common. Not at all infrequently there is some jaundice. The want of aeration of the blood is shown by a cyanotic tint, and if this exists along with icterus, a curious greenish hue may be the result. The blood commonly contains an increased number of red blood corpuscles and hæmoglobin, and the spleen is enlarged. There may be some cardiac uneasiness, amounting even to actual pain, attended by palpitation, tremor, or delirium cordis. Irregularity of the rhythm and sudden alterations of the rate of pulsation are perceived by the patient from curious fluttering sensations. The pulse is almost always weakened. The vessel may be healthy, or the reverse; the pressure is usually low; the rate frequent; the rhythm irregular. The præcordia may be found on inspection to

show abnormal appearances. A feeble, heaving impulse is often to be seen over a large area, and it is sometimes impossible to determine any distinct apex beat in such a diffuse impulse. If a definite apex beat can be ascertained, it is always weaker than in health. Occasionally the enlargement of the heart may be so great as to produce a bulging of the præcordia. This, however, is admittedly rare. The cardiac dulness is always increased, and this enlargement is necessarily in direct proportion to the amount of dilatation. The dulness is increased both to the right and to the left, but in cases of dilatation chiefly affecting either side of the heart, that side which corresponds to the cavities most involved shows the greater increase of dulness. The dulness never shows any approach to that characteristic form assumed when there is much pericardial effusion. On auscultation the sounds of the heart are found not only to be weakened in intensity, but altered in character. The first sound, more particularly, becomes shorter in duration and somewhat higher in pitch. It presents, therefore, a greater similarity to the second sound than is the case in health; and, as the long pause is often reduced by the acceleration of the heart, there may be a difficulty in determining which is first and which is second. Not infrequently the second sound is absolutely louder than the first. The first sound not infrequently presents the characters of apparent, and the second sound of absolute, doubling. In a large proportion of cases there is a systolic murmur from regurgitation at one or other of the auriculo-ventricular orifices. A tricuspid systolic murmur is far more commonly met with than one at the apex, but a mitral systolic murmur is often present also.

There is a tendency towards passive hyperæmia and œdema of the lungs, shown by slight impairment of the percussion sound, roughness of the respiratory murmur, and moist accompaniments; sometimes dulness on percussion, and diminution or loss of the breath sounds, reveal transudation into the pleural sacs. The urine is lessened, and presents the characters of a concentrated secretion, while albumin, blood, and tube-casts may make their appearance. The integumentary tissues are often cedematous. The central nervous system

frequently suffers from the disturbance of the blood supply, and many cerebral symptoms may thus be produced.

DIAGNOSIS.—The recognition of cardiac dilatation depends upon the evidence of enlargement, along with weakness of the impulse, impairment of the sounds, and feebleness of the pulse. Seeing that dilatation is so commonly associated with hypertrophy on the one hand, or with degenerative changes on the other, the opportunity of studying cases of simple dilatation is not very often afforded; but when it is present in a comparatively simple form, without any considerable hypertrophy or structural alteration, its determination is not attended by much difficulty. A wide diffusion and weakening of the impulse, and enlargement of the dulness, associated with a first sound of sharp and high pitch, an empty condition of the arteries, and a short, weak impulse in them, may be held to be significant of simple dilatation. When hypertrophy is also present, the impulse, although widely diffused, is not weakened, the heart sounds are more blunt and at the same time sustained, while the pulse is fuller, larger, and stronger. When degenerative changes are present along with dilatation, the impulse becomes still more enfeebled, the first sound still weaker, and the arterial pulse even less distinct. In addition to hypertrophy, which will be discussed immediately, there are two conditions from which dilatation must be carefully distinguished. Simple fatty accumulation and infiltration may give rise to an enlargement of the cardiac area very like that of dilatation, with a similar diffusion of the impulse; but in fatty accumulation there is by no means the same diminution of the first sound, while the radial pulse may be full, large, and strong. It need scarcely be added that in fatty degeneration there is no necessary enlargement of the heart. In pericardial effusion there is no increased area of impulse, and the form of the area of dulness is quite different from that which is found in dilatation; effusion giving rise to the characteristic pyriform outline of dulness, while in dilatation the increased area of dulness occurs laterally. The area of impulse in pericardial effusion is smaller than that of the dulness; whereas in dilatation the impulse and the dulness are practically of equal size. Not much dependence is to be placed upon the

distribution of the sounds in most cases, yet it sometimes may be possible to determine that in cases with considerable pericardial effusion the sounds are louder at the base than over the rest of the præcordia. In cases presenting pericardial friction there can be no difficulty in diagnosis, and if præcordial bulging and obliteration of the intercostal spaces are present, along with displacement of neighbouring viscera, the diagnosis is easy.

PROGNOSIS.—In forecasting the probable future of any case of cardiac dilatation, the best basis upon which to proceed is the relation between the causes of the change and the extent of its effects. If dilatation appears to be in excess of what might reasonably be expected from the causes which have induced it, the gravity of the prognosis will be greater than in cases in which opposite conditions prevail. It must further be borne in mind, that if there be evident tendency to degenerative processes, the outlook will be the more serious. On the other hand, if dilatation is accompanied by hypertrophy, the prognosis will be less serious, and the greater the tendency to hypertrophy the less grave will it become.

TREATMENT.—In addition to the ordinary rules applicable to the management of cardiac failure in general, as regards rest and exercise, food and drink, and other general points, it is necessary in dilatation to consider the removal of the causes operative in the production of the lesion, and the treatment of its effects. If the cause should be remediable, no effort should be spared to effect this. In cases where excessive stress of any kind, whether mental or physical, has led to strain, the exertion must be at once discontinued, and a period of rest, longer or shorter, according to results, must be enjoined. If dilatation should have its origin in any condition of inanition due to causes which can be obviated, such as digestive disturbances, these are to be removed by appropriate remedies. When anæmia, from whatever cause, is the factor which has led to dilatation, it should be persistently treated until it has been removed. In cases where pulmonary affections have led to dilatation of the right side of the heart, attempts must be made to remedy them; chronic bronchial catarrh, for instance, requires to be dealt with on general principles, and the effects of emphysema reduced as far as possible. Instances



of dilatation, due either to valvular disease or to any other cardiac lesion, must be treated according to the principles previously laid down.

The results of cardiac dilatation must also be, as far as it is possible, minimised or removed. The various catarrhal conditions affecting the mucous membranes, especially that of the digestive system, may be considerably lessened by such remedies as have already been advocated in the similar effects of cardiac failure. When there is much venous stasis in internal organs, leading to passive hyperæmia and œdema, such effects should be met as have been described in previous sections. In addition to paying attention to such points, a large number of other matters, which have been referred to in connection with cardiac failure, will be found to require attention in dealing with dilatation.

The employment of the cardiac tonics is in most instances indicated, and, according to the symptoms, one or other of them may be chosen. The general principles which were laid down in the section devoted to therapeutics will be found applicable here.

In presenting a few illustrative cases, only those in which the dilatation was due to causes not due to cardiac lesions will be recorded. Reference has repeatedly been made to examples of dilatation from valvular disease and degeneration.

CASE 54. *Heart Strain*.—J. O., aged 16, schoolboy, was seen 22nd July 1897, along with Dr. Percy Henderson, his complaint being breathlessness on exertion. His family history was excellent, his father, mother, and one brother, who with himself formed the entire family, being in perfect health. His social conditions had always been of the best kind, and his previous health had left nothing to be desired. The illness for which he sought advice began a few weeks before he was seen by me, while he was training for his school sports, and it simply showed itself by want of breath when undergoing the necessary exercise. It was unattended by any sensations of giddiness, faintness, or uneasiness.

The patient seemed a healthy and well-developed boy. His complexion was healthy, and his cheeks rosy. The alimentary system had absolutely no abnormality, and, from the tint of the lips and gums, there was obviously no tendency to bloodlessness. The pulse was 86 per minute, the artery soft and yielding, the vessel moderately full, the pressure low, the pulsation regular and equal. On examining the neck and præcordia, no abnormal pulsation could be seen either when standing up

or lying down. The apex beat was found in the fifth intercostal space, 3 in. from mid-sternum. The cardiac dulness began at the upper border of the third left cartilage, and it extended  $1\frac{1}{2}$  in. to the right and  $3\frac{1}{2}$  in. to the left of the middle line. In the aortic area the sounds were healthy; they were clear, but the first sound was short, sharp, and high pitched at the apex; the second pulmonary sound was markedly accentuated, but never reduplicated in any posture, or on holding the breath. In the tricuspid area there was a loud murmur, systolic in time, and varying with the position of the patient. When standing or sitting the murmur was shrill and high pitched. When the patient lay on his back it was soft and blowing, although loud. The respiratory system showed no abnormality, with the exception of wavy respiration over the bases of both lungs, and the other systems were perfectly healthy.

There could be no doubt that this was an instance of pure cardiac dilatation from physical stress.

CASE 55. *Cardiac Dilatation*.—E. G., aged 23, student, was seen along with Dr. Basil Orr, 3rd December 1897, complaining of palpitation, especially when engaged in mental operations. His family history was good, his father and mother being in excellent health, as they had always been, and his brothers and sisters being in every respect healthy. There was, however, one interesting point. An elder brother had some years ago, during his undergraduate career, experienced symptoms closely resembling those of which the patient complained. His social surroundings were admirable, his previous history was perfectly satisfactory.

The patient was slender in build, but well developed. His skin was clear, and there was a bright tint upon each cheek. There were no digestive symptoms; the tongue was clean, but slightly tremulous; the teeth were good; the abdominal organs of normal size. The colour of the lips and gums was bright, and there was clearly no tendency towards bloodlessness. The radial artery was soft and yielding, fairly full, and of moderate pressure; the pulsation was regular and equal. The pulse rate was 116. There was considerable throbbing of the vessels of the neck, and also of the parietes in front of the heart. The apex beat was in the fifth intercostal space, 4 in. from mid-sternum. The impulse on palpation was felt to be extremely diffuse, and of a short, sharp character. On percussion, the cardiac dulness extended from the third left cartilage downwards, reaching 2 in. to the right, and  $4\frac{1}{2}$  in. to the left, of the mesial plane. On auscultation in the aortic area, no abnormality in the quality of the cardiac sounds could be made out, excepting that the first sound was somewhat short, while the long pause was somewhat abbreviated. There was a faint, soft, blowing mitral systolic murmur, propagated for a little distance in every direction; but on passing towards the sternum, after the sound had waned, it became reinforced by another murmur. In the pulmonary area, the second sound was somewhat louder than in the aortic region, yet it was not sufficiently increased to constitute a definite accentuation. The first sound was replaced here by a murmur which

became louder on passing down the sternum until the level of the fourth costal cartilage was reached, at which point it had its maximum intensity, and from which it gradually faded away in every direction. It blended, as has been mentioned, with the mitral systolic murmur; but as there were two definite points where the systolic murmur reached its highest intensity, it was obvious that there were two murmurs. There was absolutely no departure from health in regard to any other system.

In this case it was quite clear that there was simple cardiac dilatation. The cause of this dilatation was, however, not so obvious. On making diligent inquiries as to all possible factors, it was elicited that the patient had for some months been over-working himself for an examination, sitting up till far on in the morning, and rising again after very few hours' sleep. Even what he was pleased to call recreation was not of such a kind as to furnish the best results, as he had been in the habit on Saturdays of playing two or three rounds of golf on one of the most hilly of our local links, which, without sufficient regular exercise and with inadequate rest, must have done much more harm than good. The patient was treated by means of ammonium bromide and infusion of digitalis, and within a fortnight Dr. Orr reported that the pulse rate had sunk to between 60 and 70, the feelings of faintness and palpitation had disappeared—in short, that satisfactory progress was being made. A visit to Nauheim has produced complete recovery.

CASE 56. *Cardiac Dilatation*.—W. C., aged 52, army pensioner, engaged in the work of a labourer, was admitted to Ward 22 of the Royal Infirmary, 9th October 1892, complaining of dropsy and breathlessness. The patient was absolutely ignorant as regards his whole family history. His environment had been such as may be imagined in the case of a man who had enlisted while young, and had seen service in many parts of the world, during both peace and war. He had never suffered from rheumatism. He had been wounded more than once in action, and had lost one of his legs in Afghanistan. He had further suffered from syphilis.

About two months before admission, he began to suffer from breathlessness, and observed swelling of the ankle and foot; as rest did not improve his condition, he sought admission. The alimentary system presented no symptoms calling for remark, excepting the presence of a considerable degree of ascites, along with some enlargement of the liver; while the spleen was also in some degree enlarged. The radial artery was healthy, the vessel was somewhat empty, and its pressure rather low. The pulsation was extremely irregular, and the rate on admission 92. The veins of the neck were somewhat turgid, but showed no pulsation.

No abnormal appearances could be detected over the præcordia. The feeble apex beat was found to be in the fifth intercostal space, nearly five inches from mid-sternum. The right and left borders of the heart were respectively  $2\frac{1}{2}$  and  $5\frac{1}{4}$  inches from mid-sternum. The heart sounds were observed to be extremely feeble, more particularly in the mitral and tricuspid regions, where the first sound could with some difficulty be determined: the second sound was more distinct, particularly in the pulmonic area. There was some cough, accompanied by frothy serous expectoration, and physical examination of the lungs showed slight impairment of the clearness on percussion at the bases behind; while there were numerous crepitations over the whole of the lungs.

In order to meet the indications furnished by the cardiac failure, the patient was treated by means of 10 minims of tincture of digitalis and 20 grains of acetate of potash every four hours for a week. By that time auscultation showed a distinct systolic murmur in the mitral area, propagated to the axilla, and another systolic murmur of a different character and tone in the tricuspid area. On the 16th October the digitalis and acetate of potash were replaced by Easton's syrup, and by the 22nd all the murmurs had disappeared; the heart was reduced so considerably in size as to be only 2 inches to the right and  $4\frac{1}{2}$  to the left of mid-sternum, while all symptoms of cardiac failure had disappeared.

In this case the cardiac dilatation may have been in part due to syphilitic changes in the myocardium, yet since complete recovery ensued as the result of treatment designed in no way to meet the possibility of specific changes, it is unlikely that this was really the case. More probably the dilatation and failure resulted from physical stress. It is of much interest to note that the heart developed murmurs while undergoing restitution, and entirely lost them when equilibrium was regained.

### CARDIAC ANEURYSM.

Arising in consequence of such alterations of the myocardium as have been discussed, localised bulging of any cavity may occur, to which is applied the term aneurysm of the heart. So far as is known to me, the change was first observed by Galeati, but attracted little attention until the work of Corvisart, who was followed by Breschet, Thurnam, Rokitansky, and other writers. A very careful study of the condition was made by Pelvet, in which may be found copious references to the previous literature. Since the appearance of his work, further



investigations have been made by many observers, some of whom will be mentioned in the sequel.

Aneurysm of the walls of the heart is commonly divided into the great groups:—the one, termed acute or sometimes false, aneurysm, arising in consequence of acute endocarditis or acute myocarditis, under which lesions it has been already mentioned; the other, called chronic, or true cardiac aneurysm, produced in consequence of gradual changes in the walls of the heart. The few remarks which follow deal entirely with the latter affection.

Cardiac aneurysm is a somewhat rare affection, and is, for the most part, a *post-mortem* surprise to the observer.

ETIOLOGY.—The usual cause producing aneurysm of the wall of the heart is an alteration in the consistency of the myocardium. The most common change in the myocardium is chronic myocarditis. This change may be produced by any of the factors which have been previously considered, but that which is most common is some interference with the lumen of the branches of the coronary arteries, as was clearly brought into prominence by Huber in this connection. The most usual change in these vessels is arterial sclerosis followed by thrombosis. Endarteritis obliterans, however, must not be overlooked, and atheroma of the aorta at the mouths of the coronary vessels has also been observed. These vascular changes lead, as has been shown previously, to alterations in the myocardium, rendering it liable to yield when subjected to much stress. There can be no doubt that besides myomalacia produced by coronary obliteration, other changes of the muscular wall may be sufficient to produce aneurysm. Chronic changes of the kind may be set up by endocarditis, and it seems probable that, as suggested by Rendu, pericardial adhesions may have a similar effect.

MORBID ANATOMY.—Cardiac aneurysm is usually single, but two, or even three, have been described in the same heart. The usual seat of the lesion is the left ventricle, close to the apex, but cases are on record in which the change was situated higher up. The aneurysm forms a definite swelling of hemispherical shape, as may be seen in Fig. 181, from a specimen kindly placed at my disposal by Dr. Harvey Little-

john ; sometimes, however, it projects to a greater degree and forms a globular mass. Its size is not usually great, but it



FIG. 181.—Cardiac aneurysm affecting the left ventricle, which had undergone rupture.

has been described as attaining almost the size of the heart itself. The cavity has a free communication with that of the ventricle, of which, indeed, it is simply in most instances a bulging. This is shown in Fig. 182, from a specimen for which I am also indebted to Dr. Harvey Littlejohn. In rarer

cases there may be a more restricted orifice leading from the heart into the aneurysm. The wall of the aneurysm is always found to be considerably altered. It is not only thin, but its structure is greatly modified. The muscular fibres are found to have in great part disappeared, and connective tissue has taken their place. The pericardium has often been described as thickened, and adhesions have been seen. Sometimes in



FIG. 182.—Cardiac aneurysm, viewed from inside of left ventricle.

a large proportion of cases, indeed, alterations in some of the branches of the coronary arteries have been found.

**SYMPTOMS AND DIAGNOSIS.** — The clinical features of cardiac aneurysm are so indefinite that its determination during life is seldom possible. The common symptoms which have been found are breathlessness, palpitation, and præcordial uneasiness, with weak pulse, feeble impulse, enlargement of the cardiac dulness, and diminution of the intensity of the sounds. These, however, are but the features of cardiac failure, and present nothing significant of cardiac aneurysm.

A characteristic attitude has been claimed for this affection by Bucquoy. This observer asserts that patients who suffer from cardiac aneurysm have a great tendency to lean forward when sitting until the chest almost touches the knees. Paul observed in one case a diastolic murmur without aortic incompetence, which he considered due to reflux from the aneurysm into the ventricle at the time of diastole. Rendu called attention to a doubling of the second sound, caused, according to him, by a state of tension of the sac. It cannot be held that any of these observations have been of real service to us in the detection of cardiac aneurysm. The instances on record have presented the clinical appearances of cardiac failure, and after a longer or shorter course the lesion terminates life, in most cases by asystole, but in some a rupture of the heart is the cause of death.

PROGNOSIS AND TREATMENT.—Inasmuch as the determination of a probable cardiac aneurysm can only be the result of inference from uncertain premisses, it is unnecessary to dwell upon the outlook in the disease, while the treatment in most cases will probably be that of cardiac failure.

### SPONTANEOUS RUPTURE.

Laceration of the heart occurs as a result of traumatism, but lesions of this kind will be considered in a subsequent section devoted to wounds; in the present connection rupture of the heart taking place spontaneously has alone to be considered. So far as can be ascertained, the first notice of such an accident is due to Harvey. Morgagni observed several cases of this kind, and one in particular, that of a woman aged 75, in whom the rupture was associated with pronounced fatty changes in the heart. Many observers since those early days have mentioned or discussed the subject, and some of the observations will be referred to in the brief summary of spontaneous rupture which follows.

ETIOLOGY.—It may be asserted, without fear of contradiction, that spontaneous rupture never occurs in a healthy heart, and some preceding lesion of the walls has always been in existence. In all cases a degenerative process, such as



will be mentioned below, has been detected, and the degenerative changes have most commonly been the result of interference with the lumen of a branch of one of the coronary arteries, which has led by ischaemia to myomalacia cordis. In some very rare cases, embolism has led to changes of a similar character, but more rapid development. It might naturally be expected that, associated with such predisposing causes of rupture, severe muscular effort might be the determining or exciting influence, and this without doubt has sometimes been the case. It must, however, be remembered that a considerable proportion of cases have been described as occurring during the night.

MORBID ANATOMY.—By far the most common seat of rupture is in some part of the left ventricle, and more particularly in that half which terminates at the apex. Odriozola found that in 71 cases the rupture occurred 10 times near the base, 28 times in the middle portion, and 33 times near the apex of the left ventricle. The most common portion of the ventricle to give way is the anterior surface. A rupture of the posterior aspect is rare, while the left border and the apex are almost exempt. Although the left ventricle is much more commonly affected than any other part of the heart, spontaneous rupture of the wall of any cavity may occur. According to Odriozola, dealing with 132 recorded instances, the left ventricle was affected 96 times, the right 22 times, the right auricle 10 times, the left auricle twice, and the auriculo-ventricular sulcus also twice. The rupture in the recorded cases has almost always been single, but according to Letulle, 18 out of 110 recorded cases presented more than one opening.

The dimensions of the opening vary greatly, but the most ordinary size is between half an inch and an inch in length. The form of the opening is extremely diverse. When small it is usually a slit, but is less frequently rounded. When larger it is occasionally in form like a curved line, but quite commonly it presents the appearance of a cross, of the letter Y, or even of a more complicated outline. The aspect of the opening on the inner side of the wall is often described as difficult to find, hidden as it is by the fleshy columns of the

ventricle, or by clots. The external aspect, although commonly hidden by clot, is easily seen after the coagulated blood has been washed away. It is in most instances larger than the inner opening. The direction of the rupture is



Fig. 183.—Spontaneous rupture of heart, affecting the right ventricle.

sometimes straight, but it is also not infrequently oblique, and in some instances it is so sinuous that the two ends of the opening have been far from each other. The pericardium always contains a large amount of blood, usually in the form of clot. Fig. 183 gives the aspect of the external opening in

a specimen for which I am indebted to Dr. Harvey Littlejohn. The form of opening is linear.

The appearance of the myocardium on inspection with the naked eye is of interest. It is commonly paler than in health, but the pale tint is often described as broken up by numerous red patches due to hæmorrhages. Sometimes those patches are of the nature of hæmorrhagic infarct, resulting from thrombosis or embolism of a coronary branch. On microscopic examination, according to Letulle, the wall of the heart in the neighbourhood of the rupture shows habitual absence of fatty changes, while various atrophic lesions of the muscular elements, of a granular or pigmentary nature, are common. A diminution of the striation is often seen along with segmentation of the fibres, or fragmentation of the cells. An increase in the amount of fibrous tissue is also frequently determined, and it must be added that rupture is occasionally found as the termination of cardiac aneurysm. This is shown in Fig. 181, p. 714. In the larger proportion of cases the coronary arteries are diseased, endarteritis deformans and obliterans being the most common lesions, associated with thrombosis. It need hardly be added that in very many instances valvular lesions and other endocardial changes are found in connection with cardiac rupture, and, in some cases, pericardial affections also.

**SYMPTOMS.**—It is only occasionally that the clinical features attendant upon cardiac rupture are so definite as to afford sufficient data upon which a diagnosis might be founded; for the most part, the symptoms are simply those of asystole. Sometimes the accident is accompanied by severe præcordial pain—pain which may be as agonising as that of angina pectoris, and which may, like it, radiate towards the shoulder and arm of one, or other, or both sides. The pain is accompanied, or speedily followed, by paleness and coldness of the surface, and extreme weakness and irregularity of the pulse. Giddiness has often been described, and vomiting has also been frequently noticed. There is usually a speedy loss of consciousness, and the breathing rapidly terminates. The accident may be even more startlingly rapid than this, and in such cases the patient has been

described as falling down in pallid unconsciousness, to die after a few irregular respirations. Such was the case in 71 out of 100 cases of rupture analysed by Quain. On the other hand, life may be maintained for a few hours, or even a few days. Five of the 100 cases discussed by Quain lived for two days. Peter cites a case in which life was maintained for twelve days; Beadles one in which death took place after the lapse of 168 days; and Rostan has recorded a remarkable instance of cardiac rupture in which the aperture was closed by pericardial adhesion and fibrinous clots, resulting in the prolongation of life for fifteen years, at the end of which period death occurred by means of another rupture. When the patient has not perished instantly there is usually a considerable amount of præcordial distress, along with the general features of cardiac failure. When life is sufficiently prolonged to permit of a careful examination of the chest, the cardiac pulsations are extremely feeble, the area of dulness increased, and the sounds almost imperceptible.

DIAGNOSIS.—The determination of a cardiac rupture must always be difficult, and the diagnosis almost impossible. Inference alone is possible in the cases attended by instantaneous death, while in those living long enough to permit of careful examination, the clinical features are too indefinite to be of diagnostic importance. Many instances closely simulate the asystole of cardiac debility or degeneration, and the remainder may be well mistaken for severe angina pectoris. The only point of difference between the two lies in the condition of the pulse, which in rupture is extremely feeble and usually irregular, while in angina pectoris it is very commonly resistant and regular.

PROGNOSIS AND TREATMENT.—These aspects of the subject require no comment.

### CARDIAC HYPERTROPHY.

Hearts of large size appear to have been first noticed by Massa, and increased thickness of the walls was undoubtedly observed by Albertini. The first clear conception of increase in the size of the heart as the result of some obstacle is,



however, due to Mayow, who described it as occurring in mitral disease, and by Vieussens, who recorded an instance of enlargement from aortic disease. An increase in the thickness of the walls, without dilatation of the cavities, was studied by Morgagni, and from his time cardiac hypertrophy has been systematically investigated by almost every writer on cardiac disease. Bertin showed by microscopic examination that in hypertrophy there is an increase in the amount of muscular tissue, and he distinguished the three classes—into which hypertrophy of the heart was so long divided—concentric, simple, and excentric. As was mentioned in considering the method of percussion from a general standpoint, Auenbrugger employed his discovery as a means of detecting cardiac enlargement. The diagnosis of cardiac hypertrophy can scarcely, however, be regarded as having been practicable until the appearance of Laennec's work, in which he, for the first time, correctly described the modifications of the heart sounds in this condition.

ETIOLOGY.—Since hypertrophy is a natural process, intended to compensate for the effects of morbid influences, it cannot really be held, as Gowers remarks, to have any morbid predisposing causes. For the occurrence of hypertrophy there must be, in truth, rather a combination of favourable conditions, allowing the possibility of such a compensatory process. It is, therefore, more correct to inquire into the conditions which further the healthy processes leading to hypertrophy, than to enter upon a fruitless quest for predisposing causes.

The state of the general nutrition is necessarily of supreme importance. Its limits must always be to a considerable extent dependent upon individual peculiarity, impressed on every one, not merely from birth, but from the earliest development of the germ plasm. The possibilities afforded by the general nutrition are always greater in early youth than at later periods of life, during which they gradually wane. They are reduced by all conditions, whether general or local, that impair the nutritive energy of the system at large, and they may be increased by careful attention to the various means of elevating the standard of general

health. The quality of the blood is one of the most important general factors, and when the metabolic processes are adequately performed, so that a sufficient amount of the various substances required by the cardiac tissues is supplied, hypertrophy is possible.

Among the local conditions allowing the occurrence of hypertrophy, is, first and foremost, the integrity of the cardiac blood vessels. This consideration has been so fully discussed in previous chapters as to require little more notice here. The condition of the blood pressure within the coronary arteries and their ramifications is really the predominating factor permitting or preventing cardiac hypertrophy. This was observed long ago by Corvisart, and has in our own time been often emphasised. If the coronary arteries are narrowed or obstructed, hypertrophy is not seen as it is when these vessels are healthy.

The relation between the work and the repose of the heart is also of great moment. Muscle cells, as well as brain cells, require their period of rest, and the only repose the heart is allowed is the period of inaction following upon the diastole. Increased frequency in the action of the heart means diminished rest; lessened frequency, the converse. If, in any case, there is an acceleration of the contractions, the amount of rest allowed to the heart is correspondingly curtailed.

The real exciting cause of cardiac hypertrophy is increased muscular activity, and the thickness of the myocardium may be said, with approximate accuracy, to be directly proportional to the amount of work which it must do. Even from embryonic life important lessons may be learned on this subject. It has already been shown, from the researches of Gillespie and myself, that during foetal life, when the blood pressure on the two sides of the heart is practically equal, the thickness of the walls of the two ventricles is similar. This fact in itself furnishes absolute proof of the proposition that the thickness of the walls of the ventricles is exactly proportional to the work which they have to perform.

The causes operative in producing the increased activity may be such as affect the whole body at large, or such as influence the circulation alone, or, indeed, only one individual

part of the heart. In the latter case, however, sooner or later, there is an implication of almost the entire circulatory apparatus.

The particular mechanism by means of which increase of work produces muscular hypertrophy is at present unknown. It is generally recognised that blood circulates more rapidly and more thoroughly through a muscle in a state of contraction than through one in a state of rest, and it is therefore possible that the increased nourishment leads to the overgrowth. This, however, is by no means the whole truth of the matter, and there seems to be in some way a direct influence of the increased contraction on the growth of the fibre.

Rejecting the view that a simple increase of work can give rise to hypertrophy, and starting with the theory of Fick in regard to muscular tone, Horvath believes that the conditions which permit of the production of hypertrophy are present in the occurrence of muscular contraction—or a stimulus to contraction—during a state of greater extension than the normal. He therefore holds that without these two concurrent factors—greater stretching and the occurrence of contraction during its presence—there is no place for the theory of Fick. Extending this idea, he shows that a muscle can be greatly stretched, even to the point of destruction, without the conditions of Fick being present, since contraction has not occurred, and no stimulus thereto has been present; while, on the other hand, these conditions may also be absent if there be no stretching of the muscle beyond the normal when contraction takes place. In the increased stretching of the cardiac walls at the instant of systole, it need hardly be added he finds a combination such as is postulated.

Increased resistance is the most powerful agent in the production of cardiac hypertrophy. Such increase in the obstacles to be overcome may have origin in the heart itself, either externally or internally, in the arterial system, in the condition of the blood, or in the organs to which the blood is supplied.

Obstruction to the action of the heart may be altogether external to it, as in pericardial adhesions. This was observed

by Morgagni and urged by Hope. It is a matter of common observation that in adherent pericardium a degree of hypertrophy of both ventricles is present, but Wilks has found that the right ventricle undergoes greater change than the left. In a previous part of the present chapter it has been shown that dilatation is a result of external adhesions, and probably the condition of over-distension is the cause of the hypertrophy. My own observations do not enable me to pronounce any definite opinion in regard to this subject, since every case of adherent pericardium which has fallen under my notice has been accompanied by some valvular lesion.

In derangement of the internal mechanism of the heart there exists a large field for the production of hypertrophy, directly or indirectly. The general principles involved in the development of hypertrophy have been discussed in the chapter dealing with general pathology. Some of the same details connected with the evolution of local hypertrophy have also been considered under the head of diseases of the orifices and valves. It is therefore unnecessary to advert to such facts except to emphasise one or two particulars. The influence of a mechanical derangement may be entirely produced by an obstacle to the onward progress of the blood. In such cases the effect of the obstacle is felt by the chamber situated immediately behind. Thus, in aortic obstruction the left ventricle is directly implicated; in mitral obstruction the left auricle undergoes immediate effects; while in pulmonary and tricuspid obstruction the right ventricle and right auricle respectively show compensatory changes. In all such instances of retro-hypertrophy from simple obstruction, it is more likely to occur unassociated with dilatation. The probable explanation of the hypertrophy in all such cases is that the obstacle to the outflow of the blood gives rise to over-distension of the cavities, so that when contraction occurs it has not only to move a larger mass of blood, but it has to force it through an orifice smaller than the normal.

Incompetence also directly produces hypertrophy. It has already been shown that in mitral regurgitation there is almost invariably some hypertrophy of the left ventricle. It is needless to spend time reiterating what has already been



said in regard to this subject, seeing that it has already been fully explained by reference to the increased amount of blood which reaches the ventricle in consequence of the regurgitation into the auricle. In such an instance as this the hypertrophy is not attended by any considerable degree of dilatation. When regurgitation is combined with obstruction, the chamber behind the diseased orifice shows the effects in their highest expression. In combined obstructive and regurgitant lesions of the aortic orifice, there is at once a high degree of hypertrophy and dilatation.

The fact must not be overlooked that mechanical derangement, whether caused by obstacle or incompetence, produces hypertrophy indirectly: mitral lesions, for instance, almost invariably give rise to hypertrophy of the right ventricle by increasing the resistance within the pulmonary circuit. The converse, although far from being so common, does undoubtedly occur, and obstruction in the pulmonary system may lead to hypertrophy of the left ventricle.

In order to analyse in a scientific fashion the effects of valvular disease on the orifices, cavities, and walls of the heart, Hamilton has summed up the effects of his cases of heart disease in the following table:—

[TABLE.]

	Weight in oz. Avoid.	ORIFICES.				CAVITIES.		WALLS (Ma. thickness).	
		Aortic.	Mitral.	P. Art.	Tricusp.	L. V.	R. V.	L. V.	R. V.
1. Normal heart . . . . .	10-13	in. ·9-1	in. 1·2-1·4	in. 1·1-1·2	in. 1·5-1·8	in. 3-3½	in. 3½-3¾	in. ½	in. ½
2. Aortic of normal size, but incompetent . . . . .	21½	·99	1·3	1·1	1·8	4	4½	5	+ ⅓
3. Aortic constricted and incompetent . . . . .	18¾	·75	1·3	1·1	1·7	3½	3½	5	+ ⅓
4. Aortic dilated and incompetent . . . . .	22¾	1·2	1·7	1·2	2	3¾	3¾	+ ¾	+ ¼
5. Mitral constricted and incompetent . . . . .	16	·97	·86	1·1	1·7	+ 3¼	+ 3½	5	½
6. Pure dilatation of mitral . . . . .	20½	1	1·68	1·27	1·98	3¾	4	+ ½	+ ⅓
7. Aortic and mitral both constricted ; aortic competent, mitral incompetent . . . . .	12¾	·7	1	1·1	1·6	3½	3¼	½	+ ⅓
8. Aortic and mitral both constricted and both incompetent . . . . .	20¼	·8	·77	1·08	1·6	3½	3½	+ ½	+ ¼
9. Aortic of natural size, but incompetent, mitral constricted and incompetent . . . . .	20½	·97	·87	1·1	1·6	3½	+ 3½	½	+ ⅓

There is a good deal that is contradictory in these results, and this is probably due to the capriciousness, as Gairdner has termed it, of the effects of hypertrophy. Taking up lesions affecting the aortic orifice or cusps alone, it will be seen that the dilatation of the orifices, the size of the cavities, and the thickness of the walls are greatest in dilatation of the aortic orifice, and this quite agrees with the usual conception that this is the gravest of cardiac lesions. Mitral disease affords in itself an interesting contrast between obstruction and incompetence; in the former there is little, if any, change, but in the latter the right side of the heart has dilatation of the orifice, enlargement of the cavities, and thickening of the walls. Combined lesions affecting both the mitral and aortic orifices and valves have but little effect on the rest of the heart, with the sole exception of combined obstruction and incompetence at both orifices, in which case there is a change on the right side of the heart.

In connection with the effects of arterial affections it is necessary to refer to aneurysm. There has been considerable discussion throughout the whole history of modern medicine as to its influence on the left ventricle. Senac showed that hypertrophy is not an invariable consequence of aortic aneurysm. Corvisart, on the contrary, regarded hypertrophy as an almost inevitable effect. In many cases aneurysm is not associated with hypertrophy of the left ventricle, and Stokes, although admitting the occurrence, states that he found a small heart co-existing with the most violently pulsating aneurysm he had ever seen. It seems to me that the matter is not difficult to explain. In many cases of aneurysm there is a considerable degree of sclerosis throughout the whole arterial system, and when this is the case, hypertrophy of the left ventricle is almost inevitable; but aneurysm may occur in consequence of some local change in the structure of the aorta while the peripheral portion of the arterial system is almost healthy. In such a case as this there is no cause directly operative for the production of cardiac hypertrophy.

The degenerative changes which take place in the blood-vessels as a natural process in advanced life, but which sometimes occur at earlier periods, are powerful factors in the

production of hypertrophy. When elasticity fails and rigidity occurs as a consequence of the compensatory fibrosis, the amount of resistance to the onward passage of the blood is necessarily increased, and hence hypertrophy occurs. This is obviously the explanation of the interesting fact, discovered by Bizot, that the heart progressively increases with age.

An increased amount of work may be thrown upon the heart from over-filling of the blood vessels in consequence of dietetic errors. Long-continued excessive indulgence in food and drink gives rise to an invariable increase in the size of the heart. The tendency is greatly increased when, along with an excess of food, there is also a surplus of any stimulating agent, such as alcohol. This has been particularly studied in the "beer-heart" of Germany, especially by Bollinger, who found the average weight of the heart of men in Munich to be 370 gm. for 61 kgm. of body weight. The abuse of alcohol, tea, and tobacco, or their allies, must be regarded as to some extent operative in producing general cardiac hypertrophy, but the effects of these substances are seen more particularly when their employment is associated with the presence of other factors, such as over-exertion.

Increased resistance may have its origin in factors which are connected with faulty chemical relations of the blood itself, for the blood vessels under such conditions contain an excessive amount of waste products. The result of such conditions is to lessen the natural affinity between the blood and the tissues, and in this way to produce a distinct resistance to the interchange which ought to take place. In this way an increase of blood pressure is brought about. Such faulty chemical processes are present in many diathetic conditions. The most characteristic example of this type is undoubtedly lithæmia. In all such conditions, from failure in the processes of the secondary digestion, the blood contains at once an excess of normal waste products and, in some cases, substances which are altogether abnormal. The results produced by these faulty chemical substances are increased peripheral resistance and rise of blood pressure.

Obstruction to the onward passage of the blood situated in the smaller arterioles and in the capillaries furnishes one



of the most important causes of cardiac hypertrophy. The effects of such increased resistance are, for the most part, seen in local hypertrophy. An obstacle to the systemic circulation gives rise to hypertrophy of the left ventricle, while in the pulmonary circulation it affects the right ventricle.

Pregnancy entails a considerable increase in the amount of work which has to be performed by the heart, and an invariable hypertrophy, particularly of the left ventricle. Larcher must be credited with the earliest scientific observations upon this subject.

In the great majority of cases of cirrhosis of the kidney there is an enormous increase, not only in the thickness of the wall of the left ventricle, but in the size of the muscoli papillares also; this change is a true hypertrophy, the greater thickness being produced simply by an increased number of muscular fibres of perfectly normal appearance. Bright pointed out in his early writings on the disease afterwards to bear his name that cardiac hypertrophy was often found in cases of kidney disease, although no valvular lesion could be detected during life or determined after death. This great observer, in attempting to discover valid causes for the overgrowth of the cardiac muscle, was led to suggest two possible factors: on the one hand, an unwonted stimulus to the heart by an altered condition of the blood; on the other, the effect of an alteration of the blood on the peripheral arteries and capillaries, demanding more powerful action to drive the blood onward. Since his days many attempts have been made to explain the cardiac hypertrophy. Traube suggested that it was produced by the destruction of part of the secretory area of the kidney, and consequently diminished elimination of water. In this view he has been stoutly supported by Bartels. But it is to be remembered that lessening of the renal outflow causes in itself no interference with the blood pressure, for, as is well known, ligation of both the renal arteries produced no marked effect on the arterial pressure. Johnson advanced the opinion that the arterioles contracted in order to oppose the passage of blood, which, on account of imperfect elimination by the kidneys, is injurious to the organism. He moreover also

pointed out that the walls of the arterioles, not only of the kidneys but of many other tissues, undergo much thickening, which he attributed in great part to hypertrophy of their muscular fibres. It has to be borne in mind, however, that these observations have not passed unchallenged, for even observers like Ewald, who allow an increased thickness of the muscular coat, hold that this is produced by an enlargement, not a multiplication, of the fibres. And Sotnitschewsky holds that when the muscular coat is thickened the change is produced by an increased amount of fibrous tissue. It must not be forgotten, also, that Thoma has found the lumen of the renal arterioles to be dilated, even when the walls are thick. Gull and Sutton propounded the view that the thickening of the vascular walls associated with the cardiac hypertrophy was produced by a hyaline fibroid change in the adventitia and in the intima, with atrophy of the muscular coat and degeneration of the nuclei of its fibres. This view has obtained but little support from subsequent investigations, the microscopic appearances described by these observers being now known to be produced by the medium in which the specimens were prepared. Few observers of eminence have given countenance to these researches. Buhl holds that the cardiac and renal changes occur simultaneously, and attributes the hypertrophy of the heart to increased activity. According to this observer, therefore, the increased blood pressure is due to the cardiac hypertrophy. But, as was shown by Grawitz and Israel, cardiac hypertrophy in itself does not raise the arterial pressure, and part of Buhl's view must, therefore, be dismissed as untenable.

One fact must not be overlooked: in many cases of chronic disease of the kidneys that change in the arteries termed endarteritis obliterans is found to be present. It must be remembered that high arterial pressure and cardiac hypertrophy may occur in other conditions than chronic disease of the kidneys. These symptoms are seen in acute nephritis and even in surgical kidney. Traube has described the presence of considerable hypertrophy of the heart within a very short period from the onset of the acute renal affection, and many subsequent observers have corroborated his results.

Now, in such cases there is no question of structural changes in the arterial walls, and it is necessary to seek a cause which will be valid in regard to them as well as with respect to chronic degenerative changes.

Mahomed threw a fresh light on the subject when he pointed out that in the course of scarlatinal nephritis a rise of arterial tension sometimes precedes the appearance of albumin in the urine. In these cases there is faulty elimination by the skin; if any interference with the functions of the alimentary tract should occur, the blood pressure rises, and, unless it be averted by acting on the skin and the bowels, albuminuria follows. The occurrence of such cases led Mahomed to conclude that the capillaries are the seat of an obstruction to the onward flow of the blood. This view has received able support from the observations of Grainger Stewart and Saundby.

There can be no doubt that in many cases—possibly related to some condition analogous to the “renal inadequacy” of Clark—slight deviations from the strait hygienic road involve liability to rise of pressure and tendency to renal symptoms. In these cases it may be concluded that, on account of faulty elimination, the blood contains waste products which should not be present. The question as to whether the obstruction is in the arterioles or in the capillaries, is one not easy of solution. If in addition to containing excessive waste products—which it may do in transient affections—the blood should have further changes, such as alterations in specific gravity or modifications in the number or condition of the corpuscles, which may occur in more permanent conditions, there will be a greater tendency to the production of cardio-vascular changes. Hamilton has advanced the ingenious explanation that a peripheral obstacle may be produced by diminished specific gravity alone. He holds that this allows the red corpuscles to mix with the leucocytes, which under ordinary circumstances lie close to the walls, and thus to increase friction. This view receives support from what we know to occur in anæmia. In a large proportion of patients suffering from anæmia, especially in cases of chlorosis, but not in these alone, the pulse pressure is high, and there may even be some

hypertrophy of the left ventricle. In other cases, however, there is low pressure with dilatation. How are we to reconcile such apparently contradictory facts? It seems to me that the cause of the difference is not far to seek. In some the condition as to nutrition is tolerably good, and the cardiac muscle is fairly nourished; in others the general nutrition is notably impaired, and the heart suffers. It would be wrong to pass from this subject without insisting upon other blood changes than mere alterations in density. There are important modifications of the red corpuscles in cases of anæmia, and Cuffer has, within recent years, laid great stress upon even greater changes occurring in renal disease. Such alterations must of necessity impair the usefulness of the corpuscles, and lessen the affinity of the tissues for them.

The great point, however, is the mode of causation of the hypertrophy of the left ventricle. Bright left the question open whether the cause lay in augmented cardiac stimuli or in increased peripheral resistance. We have seen that most observers since his time have espoused the view that the cause lay in some form of increased resistance, but that they have differed as to the exact nature and seat of the obstacle. In fact, Buhl is the only writer who now holds an opinion resembling the first alternative suggested by Bright. It appears unlikely that effete substances should cause a stimulant effect on the heart, and yet it would, for certain reasons, be unscientific to deny the possibility of such an occurrence. We must admit that impure blood may contain some substance which will act as a cardiac stimulant. Some observations lately made in Edinburgh by Craufurd Dunlop, but not yet published, have shown that when a certain proportion of urine is mixed with the blood circulating in the frog-heart apparatus, the organ is stimulated to very powerful contractions. Hence we must not deny the possibility of Bright's first alternative, but wait for further information.

There can be no doubt that the most potent cause of the hypertrophy lies in peripheral resistance, produced as already seen. The teaching of Cohnheim on this, as on every other subject which he touched, is full of suggestion. Holding that the activity of the renal circulation depends not on the want



of blood by the kidneys, but on the amount of material for secretion contained in the blood, he was necessarily led to the conclusion that, if any structural change occurred by which part of the renal secretory apparatus was destroyed, greater pressure would be necessary, and this could be caused only by increased energy of the left ventricle and a correspondingly enhanced arterial resistance. According to this view, the hypertrophy of the left ventricle is compensatory.

The researches of Hamilton on this subject are embodied in the two following tables; they certainly do not bear out the dictum of Senator.

## GLOMERULO-NEPHRITIS.

Weight.	Diameters of Orifices.				Cavities.		Walls.	
	Aortic.	Mitral.	Pulmonic.	Tricuspid.	Left.	Right.	Left.	Right.
39 oz.	·9 in.	1·4 in.	1 in.	1·5 in.	3¼ in.	3¾ in.	1 in.	½ in.

## INTERSTITIAL NEPHRITIS.

Weight.	Diameters of Orifices.				Cavities.		Walls.	
	Aortic.	Mitral.	Pulmonic.	Tricuspid.	Left.	Right.	Left.	Right.
+18½ oz.	·9 in.	1·2 in.	1·1 in.	1·7 in.	3⅔ in.	3⅝ in.	+¾ in.	+⅓ in.

Some affections of the lungs are almost invariably associated with hypertrophy of the right ventricle. Gairdner long ago emphasised this fact. Emphysema presents the most remarkable instance of such association. In consequence of the destruction of a large proportion of the blood vessels, and constriction of those which remain, there is much obstruction of the blood current, leading invariably to right-sided hypertrophy. Chronic interstitial pneumonia produces similar effects. Although cardiac hypertrophy is altogether absent in the overwhelming proportion of cases of phthisis pulmonalis, it is by no means infrequent in that variety commonly known

as fibroid phthisis. The results which Hamilton has obtained are contained in the tables which follow :—

## STONEMASON'S LUNG.

Weight.	Diameters of Orifices.				Cavities.		Walls. (Max. Thickness.)	
	Aortic.	Mitral.	P. Art.	Tricusp.	L. Vent.	R. Vent.	L. Vent.	R. Vent.
+ 10½ oz.	1 in.	1·3 in.	1 in.	1·7 in.	3½ in.	3¼ in.	+ ⅜ in.	⅓ in.

## CHRONIC BRONCHITIS AND EMPHYSEMA.

Weight.	Diameters of Orifices.				Cavities.		Walls. (Max. Thickness.)	
	Aortic.	Mitral.	P. Art.	Tricusp.	L. Vent.	R. Vent.	L. Vent.	R. Vent.
+ 15½ oz.	1 in.	1·4 in.	1·2 in.	1·6 in.	3⅜ in.	3¼ in.	½ in.	¼ in.

## PULMONARY PHTHISIS.

Weight.	Diameters of Orifices.				Cavities.		Walls. (Max. Thickness.)	
	Aortic.	Mitral.	P. Art.	Tricusp.	L. Vent.	R. Vent.	L. Vent.	R. Vent.
+ 9¼ oz.	·9 in.	1·2 in.	1·1 in.	1·6 in.	+ 2½ in.	+ 3⅛ in.	⅜ in.	⅓ in.

## CHRONIC INTERSTITIAL PNEUMONIA.

Weight.	Diameters of Orifices.				Cavities.		Walls. (Max. Thickness.)	
	Aortic.	Mitral.	P. Art.	Tricusp.	L. Vent.	R. Vent.	L. Vent.	R. Vent.
11½ oz.	9 in.	1·2 in.	1·1 in.	1·6 in.	3½ in.	3¼ in.	+ ⅜ in.	+ ⅓ in.

General hypertrophy of the heart may have its origin in simple over-action; this is one form of what has been

called by Cohnheim idiopathic hypertrophy. The increased action may be the result of several different factors. Excessive physical exertion has, since the writings of Senac, been well known as a cause. The heart in those who are engaged in severe muscular effort undergoes hypertrophy when the nutritive possibilities of the heart are such as to allow of it. Allbutt, Myers, and Fothergill in this country, Da Costa in America, and Seitz, Fräntzel, and Laache on the Continent, have ably urged this fact. Such a result is easy to understand. Increased muscular exertion invariably produces augmentation of the rate of the blood flow, necessitating an acceleration of the heart, and probably an increase in its force. Very little reflection is necessary in order to arrive at the expectation that an increase in the rate and the force of the heart will further augment the rapidity of the whole circulation, and increase the amount of work which the heart has to perform. Observation proves the correctness of such expectation, for the arteries show an increase of blood pressure even when reflex dilatation of the arterioles has occurred with cutaneous hyperæmia and copious perspiration.

Long-continued emotional disturbance gives rise also to general hypertrophy. It apparently acts in a manner analogous to the production of cardiac hypertrophy through muscular exertion. The increased rate of pulsation gives rise, through augmented aspiration and expulsion, to an increased arterial pressure leading to hypertrophy, hence the simple palpitation accompanying mental excitement passes directly into hypertrophy.

Simple increase in the rate of the heart, apparently produced through the agency of the nervous system, may be found in certain nervous affections, but above all others in Graves' disease. In such cases of nervous affection the general rule is that hypertrophy makes its appearance, but this rule is subject to certain exceptions according to the general condition of nutrition.

**MORBID ANATOMY.**—Hypertrophy causes an increase in the size and weight of the heart, either in whole or in part. The affection may therefore be regarded as sometimes general and sometimes local. While it is rare for every part of the

heart to undergo enlargement, it is usual to find that several of its cavities and their walls are implicated.

An increase in weight is the most certain test of cardiac hypertrophy. Attempts to ascertain the normal weight of the heart are mentioned by older writers, but the first accurate methods by directly weighing the organ are to be found in the work of Bouillaud. Bizot measured the thickness of the walls of the cavities, and in this way established accurate standards as regards size. By the labours of Clendinning, Reid, Ranking, Peacock, and Boyd, a great amount of useful information has been collected in regard to the normal size and weight of the heart, and this has again been more recently revised by Hamilton. The normal weight of the heart has already been stated in the anatomical introduction, and the measurements of the individual portions have also been fully considered.

The standard of the myocardium, estimated according to its weight, must only be accepted on the understanding that the weight of the heart, although an excellent index of its size, has no relation to its powers. The results require to be corrected by reference to the size of the cavities, the thickness of the walls, and the integrity of the structure.

It must be obvious also that in order to obtain an accurate estimate in regard to the weight and size of the heart, the facts as to the age, the sex, the height, and the weight of the individual must be borne in mind, as well as the circumstances under which death has taken place, and the truth in respect of the healthy heart can only be ascertained in the case of those who have died suddenly from traumatic causes while in health.

General is much less frequently met with than local hypertrophy. In the latter variety, however, the change is seldom entirely limited to one part of the heart; other portions take part, at least to some extent, in the hypertrophy. The order of frequency in which the different parts of the heart are affected is, left ventricle, right ventricle, left auricle, right auricle.

The form of the heart undergoes considerable changes, and these differ according to the part mostly affected. Auricular hypertrophy never causes any change in the form of the heart,



and only in cases of ventricular hypertrophy can a change in form be seen. In hypertrophy of the left ventricle the apical portion of the heart is increased and the entire heart is elongated, while in hypertrophy of the right ventricle the apex becomes blunter and the whole of the heart broader and rounder.

An instance of increase in the weight of the heart without any apparent cause, to be regarded therefore as a possible example of the idiopathic hypertrophy of Cohnheim, was recorded by Peacock, in which the heart weighed 40 oz. In the increase produced by local causes connected with the aortic orifice Van der Byl found a heart weighing 36 oz., and Bristowe recorded a similar instance in which the heart weighed  $46\frac{1}{2}$  oz. In combined aortic and mitral disease, Peacock mentions hearts weighing  $21\frac{1}{2}$  oz. in men and 23 oz. in women. In affections of the pulmonary orifice of congenital origin, Peacock observed weights of 12 oz. in men and  $17\frac{1}{2}$  oz. in women.

An increase in the thickness of the walls is almost invariable, yet the fact must not be overlooked that if hypertrophy be associated with dilatation, the thickness of the walls may be very slightly, or even not at all, increased. As hypertrophy is usually present in a greater ratio than dilatation, there is, nevertheless, almost invariably an increased thickness of the wall in spite of the latter. The usual thickness has already been stated in the chapter devoted to anatomical facts, but it may be recalled that the thickness of the wall of the left ventricle is on an average about  $\frac{1}{4}$  in. at the apex, and  $\frac{1}{2}$  in. at the base. If the wall of the left ventricle be three-fifths of an inch thick, it is to be regarded as hypertrophied. The right ventricle is usually  $\frac{1}{8}$  in. thick in health, but in hypertrophy it may reach one-third or even half an inch in thickness. Cases are on record in which it has been very much more than this, but these are rare. The usual thickness of the left auricle is about  $1\frac{1}{2}$  lines, and in hypertrophy it may attain to 3 lines. The right auricle rarely exceeds 1 line in health, and when hypertrophied it may reach a thickness of 2 lines. The dimensions of the walls, according to Letulle, are as follows:—

	Normal.	Hypertrophied.
Right ventricle .	5 to 6 millimetres.	8 to 10 millimetres.
Left ventricle .	10 to 15     ,,	18 to 30     ,,
Right auricle .	2 to 3     ,,	4 to 5     ,,
Left auricle .	2 to 3     ,,	5 to 6     ,,
Ventricular septum	12 to 15     ,,	16 to 25     ,,

These figures are much smaller than those given by some other observers, and Letulle points out with justice that many of the results on record have been obtained by want of care in measurement. In order to obtain reliable observations the walls

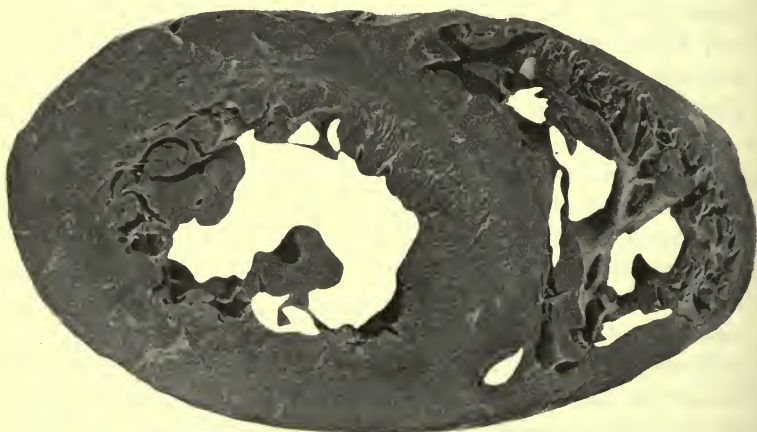


FIG. 184.—Hypertrophy of the left ventricle in chronic renal disease.

themselves must be carefully measured, and the epicardium and columnæ carneæ must not be included. An example of hypertrophy of the left ventricle is shown in Fig. 184, and several examples are shown in other parts of the work.

It is perhaps scarcely necessary in this age to refer to the old classification of Bertin, by which hypertrophy was divided into the three varieties of excentric, simple, and concentric, according as the cavities were enlarged, normal, or reduced in size. Concentric hypertrophy received its death-blow at the hands of Cruveilhier, and simple hypertrophy must be regarded as being very rare indeed. The real fact is that all hypertrophy is attended by some dilatation; it may indeed be

small, but it is constant, and it is impossible to differ from the candid statement of Letulle that he has never met with an example of simple hypertrophy. The whole question lies in a nutshell. Differences in the size of the cavities depend on the phase in which the heart comes to still-stand. If a hypertrophied heart, nearly simple in type, is found at a post-mortem examination in the final state of systole, it may seem to be like an instance of concentric hypertrophy. A little reasoning, however, will show that concentric hypertrophy is impossible. At the termination of the expulsive phase of the systole, as was shown previously, the cavities of the heart are practically obliterated with the exception of a small wedge-shaped space between the auriculo-ventricular and arterial cusps. It is impossible to conceive of any condition in which the cavities can be rendered smaller than they naturally are at this stage, and concentric hypertrophy must therefore be regarded as a vain imagination. Simple hypertrophy is indeed possible, not merely as a speculative opinion but as a reasonable expectation. If it ever really occurs it must, nevertheless, be uncommonly rare. It is interesting to observe that the views of Bertin have never been altogether left without witness. Horvath, for example, believes in the existence of concentric hypertrophy, and he finds in the theory of Fick an explanation of the incidence of Bertin's different forms of hypertrophy. In excentric hypertrophy the heart during life and after death has the impress of over-extension; in simple hypertrophy the muscle during contraction resumes its original length; in concentric hypertrophy, on the contrary, the stretched muscle during the stimulus to contraction does not only regain its former length but remains shorter.

The structure of the heart shows considerable alteration in hypertrophy. The muscle is firmer than in health, but the cut section does not, as a rule, show any change in appearance, apart from the alteration in thickness, although it is occasionally somewhat darker than in health. If degenerative processes are associated with the hypertrophy, as is often found, the colour may be somewhat paler and the texture softer than in health. Such degenerative changes are far



from uncommon, but Hamilton is undoubtedly correct in stating that they have been much exaggerated.

Meigs has arrived at the conclusion that compensatory hypertrophy, as ordinarily described and understood, has no existence; that all hypertrophied hearts are degenerated and enfeebled. He allows that this position will be very difficult to establish on account of the prejudice, as he terms it, which exists in favour of old beliefs of long acceptance. He grants it to be almost certain that in hypertrophy the number of muscular fibres is increased, but he will not allow that this increase in size is attended by increase of strength. Meigs takes up the position that the appearance of hypertrophied hearts proves that the muscular tissue is always diseased, and concludes that if the enlarged heart be acknowledged to be always degenerated and weakened, there can be no escape from accepting his view that compensatory hypertrophy is a myth.

This author believes that regurgitation does not always cause hypertrophy, and he asserts that if this can be proved, the theories with regard to compensation are rendered untenable. He is of opinion that the doctrine of an increase in the size of a muscle when called on to perform severe labour has been pushed much farther than the facts justify, and suggests that a muscle improves in efficiency much more than it increases in bulk and weight; he asserts that no development of the muscles in an athlete or a labourer is comparable to the increase in size and weight of the heart in hypertrophy, which is said to be compensatory and conservative. Another consideration which he urges is, that no demonstration has ever been given of increased arterial pressure in the case of cardiac hypertrophy. The views of this author are so intermingled with faulty observation that his conclusions cannot be regarded as resting on safe premises.

The microscopic appearances of the heart in hypertrophy have been much discussed, the point which has given rise to most controversy being a difference in opinion whether the increase in the size and weight of the heart is due to an enlargement of the fibres, or an increase in their number. The observations of Hepp led him to the conclusion, based upon



careful measurement, that the average thickness of the fibres in hypertrophy was greater than in health. It cannot be said that his statements are such as to excite confidence in any one who takes the trouble to peruse them, since he asserts that the fibres are sometimes four times as thick as in health. Zielonko, on the other hand, concluded from a large number of observations that the average size of the fibres in hypertrophy was somewhat less than the average in health. The most recent descriptions of the condition are those furnished by Letulle, who, with considerable caution, ranges himself on the side of those who believe that the individual elements undergo an increase in size.

In many instances of hypertrophy the microscope fails to reveal any divergence from the healthy standard. The determination of a possible increase in the diameter of the fibres, or augmentation of their number, is so beset with difficulties as to be, if not impracticable, at least uncertain. This aspect

of the subject, accordingly, requires no further remark. In the larger proportion of hearts examined, the muscular tissue is found to be the seat of some processes in part formative, in part regressive. Amongst the muscular bundles, and even between the individual fibres, may be seen newly formed fibrous tissue containing many nuclei. This is the result of some interstitial myocarditis. At the same time, retrograde changes may be detected in the muscle cells. Some aggre-

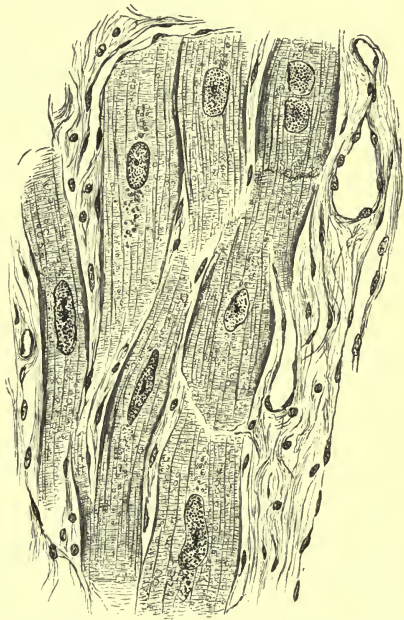


FIG. 185.—Section longitudinally through the left ventricle of an old-standing case of hypertrophy,  $\times 300$ . It shows some pigmentary and granular changes of the fibres, along with some increase of the connective tissue between and amongst the bundles.

gation of pigment is common at the nuclear poles, but of more significance is the presence of granules in the cells. Without doubt these changes are the expression of long-continued strain, and it cannot be matter for surprise that they should often be seen; it is clear that most hypertrophied hearts are examined when cardiac failure has followed the ultimate possibilities of the compensatory process. The illustration, Fig. 185, which is taken from an instance of long standing, shows these changes very distinctly.

SYMPTOMS.—It is by no means an easy task to determine which of the many clinical appearances commonly associated with cardiac hypertrophy are really produced by it. A very large number of symptoms which have been, and are still sometimes, credited to it, are in no sense to be regarded as the result of hypertrophy, but are really effects of the previous morbid conditions from which it has arisen.

There may be some cardiac uneasiness and even attacks of anginal character. It is a mistake to say, as Walshe does, that pain is rare in hypertrophy. Its presence or absence is really conditioned by the primary affection to which the hypertrophy is consecutive. The patient may suffer from various subjective sensations connected with the head, more especially pain in the head, singing in the ears, and flashes before the eyes. In many instances, however, there is absolutely no trace of such symptoms. Breathlessness is never a prominent symptom when hypertrophy is adequate. Dyspnoea on exertion, however, is by no means infrequent, seeing that hypertrophy is rarely sufficient to compensate for the original disease.

The appearance of the patient presents nothing, in most instances, that is characteristic. In certain cases, however, there is a high colour upon the cheeks, the temporal vessels stand out distinctly, and the carotids heave violently. These symptoms are caused by various factors.

The pulse reveals different characters according to the primary affection; but, speaking generally, it may be stated that the vessel is apt to be full, and its pressure high, while the rate has a tendency to be reduced. It is usually regular and equal. Besides the characters which are impressed upon it by the original affection, it must be remembered

that if hypertrophy is followed, as is so commonly the case, by fatty or fibroid changes, the state of the pulse will undergo modifications accordingly.

There may be some bulging of the region to the left of the sternum, with an increased width of the intercostal spaces. The area of impulse is enlarged, and a diffuse beat may be seen in several interspaces. On palpation the increased energy of the pulsation can be thoroughly appreciated, but it will be found that along with the greater intensity and wider diffusion of the impulse there is still a distinct apex beat. Perhaps the most characteristic change in the impulse is that it is slower in its occurrence and longer in its duration than in health. The term "heaving" is therefore frequently applied to it. There is frequently another characteristic of some importance, the apex beat giving a quivering sensation to the hand. It is not to be described as vibratory or thrilling, but really gives more a feeling of shuddering. It is, therefore, entirely different on analysis from the thrill which accompanies so many endocardial murmurs. When there is much dilatation along with hypertrophy, the impact is very different from that which has just been described, as it is then, although forcible, often extremely short in duration. There is more or less enlargement of the heart, the area being increased both transversely and from above downwards. The cardiac sounds are considerably modified. In many instances it is, on account of the original affection, rather difficult to ascertain what changes are produced by the hypertrophy, but judging from that which occurs in the course of chronic nephritis, for instance, it is possible to determine the changes due to the compensatory process. These are in the first instance confined to the left ventricle. The first sound becomes lower in tone and longer in duration, but, if there is much dilatation, the sound is correspondingly sharper in character and shorter in duration. These changes are more especially to be observed at the apex, and along the left edge of the præcordia. The first sound in the tricuspid region may present no alteration. In many instances there is an apparent doubling of the first sound, and this can be heard most distinctly over the tricuspid region. In the aortic region, the second sound is considerably



accentuated as compared with the pulmonary, and there is occasionally a doubling of the second sound, in which the aortic portion may be proved to lag behind the pulmonary.

Such symptoms as venous stasis, cedema, and cyanosis may be found. They are, however, not the consequence of hypertrophy, but the effects of inadequate compensation.

Hypertrophy of the left ventricle is almost always associated with increased arterial pressure, and it seems probable that, as Kirkes pointed out, this may in itself tend towards degenerative changes in the arteries. It must not, however, be overlooked that such degeneration of the arteries is the result of conditions antecedent to the occurrence of hypertrophy, and that it probably has been blamed for effects which were coincident with, but not resulting from, its development. Even allowing this, it must be admitted that hypertrophy of the heart by increased arterial pressure tends through stress to cause arterial degeneration. The incidence of hypertrophy has undeniably a tendency to induce rupture of diseased vessels, and the statistics collected by Quain show that in sixty-five cases of apoplexy there was cardiac hypertrophy in two-thirds.

The left auricle rarely gives any direct evidence of hypertrophy. Some authors speak of relative dulness in the second left intercostal space, and of a distinct impulse preceding the apex beat. Such appearances must be extremely rare, and the only means of determining whether hypertrophy of the left auricle is present or not is by indirect evidence. The loudness of the presystolic murmur might, on the current view of its production, be held to yield indications as to the condition of the auricle, but we do not at present understand the reason for the remarkable variability of the murmur, and this lack of information interferes with its being accepted as in any sense a reliable guide.

Hypertrophy of the right ventricle is a means of preventing the appearance of many consequences of implication of the right side of the heart. It is the great means whereby venous stasis and its train of serious symptoms are obviated or lessened. When right-sided hypertrophy is well developed there may be no sign of venous engorgement, save in the lungs in those cases having for their origin some



mitral lesion. The symptoms of which the patient complains are for the most part those of the primary lesion, such as breathlessness and uncomfortable sensations connected with the action of the heart. There is no change in the radial pulse belonging to the condition. There may be a considerable amount of venous pulsation in the neck, usually of auricular, but sometimes also of ventricular rhythm. The præcordia may show some bulging of the lower sternal and epigastric regions, and there may be very obvious pulsation in this area. The apex beat is often invisible, but when present it usually extends farther to the left than in health. The reason for this disappearance is the enlargement of the right ventricle, which, as it were, overlaps the left, and thus prevents it from being so close to the parietes as in health. Palpation confirms these observations, and also reveals the fact that the apex beat is enfeebled. The area of cardiac dulness is enlarged, both to the right and left, but it is not, as is the case in regard to the left ventricle, elongated downwards. Auscultation shows that there is but little alteration in the sounds of the heart, excepting that the pulmonary second sound is commonly accentuated, and often doubled, both of which appearances usually result from the lesion which has led to the hypertrophy.

Enlargement of the right auricle is much more easily ascertained than is the case in regard to the left, but this enlargement is not commonly produced by hypertrophy. It may reveal itself by means of dulness to the right of the sternum in the third and fourth interspaces, and an impulse occurring before the apex beat.

DIAGNOSIS.—The only reliable means by which hypertrophy of any part of the heart may be recognised lies in the increased force with which that part of the heart is acting. As regards the left ventricle, the increased energy and heaving nature of the impulse, along with the deeper pitch of the ventricular sound, if this is not obscured by some murmur, should be sufficient to effect a diagnosis. There are no reliable means of establishing a diagnosis of hypertrophy of the left auricle. Hypertrophy of the right ventricle and auricle may give rise to impulses which in health are not present. In

addition to these facts, the alteration in the area of cardiac dulness will be of service, and modifications of the radial pulse may be found when the left ventricle is implicated. From dilatation, hypertrophy may be distinguished by the character of the impulse and sounds, as well as by the nature of the pulse in the case of the left ventricle. As, however, the two conditions are frequently associated, the question rather comes to be whether hypertrophy or dilatation is in the ascendant. This can only be determined by carefully weighing the evidence furnished by physical examination.

From displacement of the heart, as well as an uncovered condition, resulting from retraction or collapse of the lungs, hypertrophy is to be differentiated by the altered characters of impulse and sounds already fully described.

Increased activity of the cardiac contraction, such as is commonly found in palpitation, might possibly be erroneously taken for hypertrophy, but on careful examination it will be found that the impulse is short and sharp, while the sounds are high-pitched and ringing, and the cardiac area is not increased. Pericardial effusion is usually regarded as a possible source of error in diagnosis, but along with an increase in the area of dulness, and in some cases a distinct prominence of the præcordia, there is diminution of the impulse and of the sounds. It is unnecessary to add that the mode of development of the symptoms is often in itself such as to lead to correct conclusions.

PROGNOSIS.—Seeing that hypertrophy is a compensatory, if not conservative, process, it would be absurd to enter upon a speculative discussion of any general matters connected with its prognosis. There are, nevertheless, some questions connected with this subject which require consideration. One important point is concerned with the presence or absence of such nutritive possibilities as will permit the development of hypertrophy sufficient to compensate for the primary disease. Another consideration of vital interest is to determine whether the nature of the primary disease admits of adequate compensation by means of hypertrophy. A further matter requiring attention is to ascertain if a moderate degree of hypertrophy can restore the disturbed equilibrium, or if an

excessive degree of hypertrophy is required. Each of these considerations requires in every instance to be most carefully discussed.

If it seems probable that the nutritive possibilities will permit the development of adequate hypertrophy; if such hypertrophy is apparently equal to meet the demands made upon it; and if a moderate development of hypertrophy appears sufficient to restore the balance, the prognosis will be satisfactory: but if the cardiac nutrition is insufficient; if compensation can only with difficulty be maintained; or if an excessive hypertrophy is required, the outlook is more serious. The two former conditions are easy of apprehension, and it can without difficulty be understood that when great hypertrophy is required, and is developed, the heart more rapidly outgrows the nutritive possibilities of the coronary arteries.

One great difficulty in attempting to forecast the future in any given case arises from uncertainty in determining the presence of concomitant conditions. The extent of the dilatation which almost invariably accompanies hypertrophy, and the presence or absence of degenerative changes in the myocardium, must always render the prognosis somewhat indefinite.

All these different aspects of prognosis depend upon the causes leading to hypertrophy, and when these are serious and permanent the prognosis is necessarily more grave. To this part of the subject it is scarcely necessary to devote any more attention, seeing that the gravity of the different lesions leading to hypertrophy is considered in the different chapters to which these causes belong.

During the last, and even in the earlier portion of the present century, much discussion was devoted to the possible harmfulness of hypertrophy. Most of the fears expressed in regard to it have been entirely set at rest by a more thorough understanding of the nature of the process, and the only survival of such questions, that can be regarded as in the least degree justifiable, is the apprehension that when in the arterial walls rigidity has taken the place of elasticity, hypertrophy of the heart may lead to a rupture of the arteries. This is by no means infrequent in renal cirrhosis with arterial sclerosis.

Even in such an extreme case it must be admitted that the power of adaptation possessed by the heart sometimes leads to a lessening of its energy, by which the blood pressure is reduced. In the arterial sclerosis of elderly people, the heart usually begins to fail when the vessels become rigid beyond a certain point, and this event, which must be regarded as another method of compensation, averts the danger of rupture, although it brings new evils in its train.

There is another consideration which demands attention. The question is often asked whether a cardiac hypertrophy once developed ever disappears. It seems undoubted that it does disappear whenever the cause which has led to it is removed. The hypertrophy of the left ventricle, which is present in subacute nephritis, almost invariably passes away with a return to healthy conditions in the kidney, and, if this be so, there can be no manner of doubt that a similar disappearance of hypertrophy can, and probably does, always take place on the removal of the cause.

TREATMENT.—As cardiac hypertrophy is absolutely beneficial, and probably never exceeds the extent demanded by the circumstances in which it takes its origin, it does not in itself require treatment. As the hypertrophy may pass the limits of nutrition, and thus give way to degenerative changes, it is always necessary to place the circulation in such favourable conditions as will prevent an excessive development of the compensatory change. This fact was well recognised by Senac, and is particularly emphasised in his remarks upon this subject. With this end in view, it is therefore necessary, as far as possible, to lessen the stress under which the circulation is carried out, and also to diminish the frequency of the heart beat, so as to permit the longest possible period of repose. The general nutrition of the system must be well sustained, or, instead of hypertrophy, dilatation is developed. The alimentary processes require careful watching, not merely as regards the adequacy of digestion, absorption, and elimination, but to see that there is no distension of the hollow viscera within the abdomen. The functions of the skin and of the kidneys should also be maintained. If there should be any appearance of cardiac failure, the special drugs applicable to this condition



will be required, more particularly digitalis or strophanthus. The question of stimulants comes to the front in every case, and although they are unnecessary under ordinary circumstances, they are nevertheless imperatively demanded when the heart threatens to fail. If there is evidence of too much arterial pressure, iodide of potassium or nitro-glycerine may be employed. When any disagreeable symptoms, such as cardiac uneasiness, become manifest, they must be met by the adoption of such measures as will be discussed in the following chapter.

## CHRONIC INFECTIVE PROCESSES.

There are two chronic infective diseases which affect the myocardium—tubercle and syphilis. These two affections are of very different degrees of frequency.

### TUBERCLE.

In an acute miliary tuberculosis, it is quite common to find grey tubercles in the myocardium. It sometimes, although rarely, happens that larger tubercular deposits in the form of caseating masses are seen. In neither of these cases are any characteristic features present during life, and both forms occur as a post-mortem surprise. Tubercle, therefore, plays but an insignificant part in the morbid anatomy of the muscle, as compared with the serous membranes of the heart.

### SYPHILIS.

Leaving the conception of Corvisart, that vegetations were gummata, as a matter now solely of historic interest, the first recognition of cardiac syphilis is due to Ricord. The real study of this condition, however, began with the observations of Virchow. Since the appearance of his work on the subject, a considerable number of cases have been recorded, not only in our own country but in France, Germany, and Italy, from which some definite conclusions can be drawn.

ETIOLOGY.—The implication of the myocardium belongs almost exclusively to the tertiary period, and, according to

Jullien, the average period elapsing between primary infection and cardiac implication is ten years, the extremes varying between one year and eighteen years. The age of those affected has also differed greatly, but, as might be expected, the period of middle life has been most fruitful in the production of examples. The male sex must be credited, as Petit states, with two-thirds of the cases. Hereditary syphilis has in several undoubted cases been the cause of myocardial disease, as has been, for example, shown by Coupland.

MORBID ANATOMY.—All observers agree that the seat of election in syphilitic changes is the left ventricle; the right ventricle is rarely implicated, and the auricles only exceptionally.

The characteristic lesion is the gumma, which may attain very different sizes. The mass is sometimes so deeply placed in the thickness of the wall as to escape detection except on section, but perhaps more commonly it projects outwards or inwards. If such be the case, there is always some thickening of the epicardium or endocardium, and when the former structure is involved there is commonly some adhesion of the pericardium. In association with the gummatous deposit there is almost invariably some fibroid change in the myocardium. This chronic myocarditis usually serves to isolate the gumma, but, as in a case narrated by Newton Pitt, softening may ensue and result in an aneurysmal change. There is, as a rule, but one gumma but there may be several.

It is still debated whether syphilis may produce a chronic myocarditis in the absence of gummata. Notwithstanding the weighty opinion of Jullien to the contrary, it must be allowed that, as Mraček states, patches of fibrous tissue occur in connection with endarteritis obliterans. Such localised changes show atrophic processes in the muscular fibres, with the appearance of pale fibrous areas. The unaffected fibres are said to be enlarged, but to be also frequently affected by fatty degeneration.

SYMPTOMS.—Myocardial syphilis often remains perfectly latent until the patient dies suddenly from asystole. This is by no means, however, to be regarded as a general rule, and pain, resembling the classical features of angina pectoris, may

be experienced. This has been insisted upon by Pearce Gould, as well as Hallopeau, and Phillips has more recently called attention to this symptom. Severe dyspnœa has often been observed, and is emphasised by Jullien as a prominent symptom. Extreme frequency of the pulse has been more particularly described by Semmola. The usual symptoms have been those of cardiac failure, with weakness and irregularity of the pulse, enlargement of the heart, and feebleness of its impulse and sounds. The common termination is brought about by asystole.

DIAGNOSIS.—The recognition of syphilis of the myocardium is beset by considerable difficulty on account of the want of definition in its symptoms. The disease may be suspected if, with other evidence of constitutional syphilis, there is any cardiac weakness without other obvious cause for it, more especially if there should be an amelioration of the symptoms on the adoption of antisyphilitic remedies.

PROGNOSIS.—The outlook is always serious, and sudden death is very common. In the great majority of cases on record, the affection has not been suspected until the inevitable termination has been at hand. Some instances of apparent recovery have been described by Lancereaux and other observers.

TREATMENT.—The management of myocardial syphilis is necessarily based upon a thorough employment of specific remedies, along with such cardiac drugs as may be indicated. Iodide of potassium in large doses must be the principal agent employed, and it may be combined, according to circumstances, with digitalis or strophanthus. The use of mercury seems to have been of much service in many instances, administered either internally or externally.

## NEW FORMATIONS.

A very large number of new formations affecting the heart are on record. Several of these are but rarely found. Amongst the less common forms myoma, fibroma, and lipoma may be mentioned. The most frequent kind of new formation is cancerous. In 21,954 autopsies mentioned by Köhler,

Tanchon, and Willigk, there were 21 cases of cancer of the heart. It usually occurs as a secondary deposit, and is extremely rare as a primary disease. A large number of cases of the former class are on record, which have been carefully analysed in the work of Bodenheimer, but since the date of its appearance a number of other instances have been collected. As a primary affection, Petit states that only 7 cases are on record. The affection is found in any age, from infancy onwards, but it has appeared most commonly about or after the age of forty-five. Men are more subject to the affection than women. Bodenheimer's statistics, dealing with 45 cases, show that it was restricted in 7 instances to the left ventricle, in 3 to the right ventricle, and in 2 to the right auricle. In all the rest of the 45 cases there were multiple changes. The position in which it takes its origin is the connective tissue between the muscular fibres.

There is but little in the clinical features of such significance as to lead to the recognition of the disease. If not entirely latent the affection simply produces evidence of cardiac disturbance, præcordial pain, palpitation or fluttering, breathlessness, along with irregularity of pulsation, weakness of impulse, and feebleness of the heart sounds—such are the features which have most commonly been found in cases which were found ultimately to be cancer of the myocardium.

With such indefinite symptoms, it cannot be wondered that the diagnosis is somewhat uncertain. Sée is of opinion that, in the absence of valvular lesions, rheumatism, alcoholism, and other reasons for cardiac failure, cancer of the heart might be suspected if there should be a tendency to failure, in the presence of cancer elsewhere, or if there should be a cachexia suggesting the presence of carcinoma. If such a diagnosis can be established, the forecast must necessarily be of the gravest description, and the treatment merely symptomatic.

## WOUNDS OF THE HEART.

Injuries of the heart produced by wounds have attracted a great amount of attention ever since the description of their symptoms by Celsus. The means by which the localisation



of a cardiac wound might be attained was described by Fallopius, and the healing of such a wound appears to have been first described by Wolf. Senac attempted to assess the degrees of danger resulting from wounds of different parts of the heart, and Morgagni enunciated a correct principle in stating that the pressure produced within the pericardium by the blood escaping from the heart was the real cause of death. During the end of last and throughout the present century, a very large number of cases have been placed on record, of which a very considerable proportion made an absolute recovery. The elaborate monograph of Fischer furnishes an exhaustive analysis of those cases.

Wounds of the right ventricle are more common than those of the left, in the proportion of 123 to 101, while in 26 instances both ventricles were wounded. The right auricle is more than twice as commonly wounded as the left, the figures being 28 to 13. The injuries which have been recorded vary immensely in kind. The whole heart has sometimes been torn off below the great arteries, and openings of every form and size have been found in the myocardium.

The symptoms which have usually been described have necessarily varied with the extent of the injury. When a large wound has been made, the symptoms are those of collapse, with pallor and syncope. When, however, the injury is small, or if, instead of involving the muscular wall of the heart, there should be injury of one of the coronary arteries, there is a gradual development of such symptoms as paleness of the face, weakness of the pulse, with feeble cardiac impulse and sounds, occasionally attended by pain in the præcordia and faintness, as well as vomiting, convulsions, and other nervous disturbances. In some instances reaction phenomena are to be found, such as cardiac excitement, with forcible and frequent pulse.

The physical examination of the præcordia sometimes reveals an increased area of cardiac dulness, and very various auscultatory phenomena. Schrötter speaks of murmurs of a hissing or blowing character, occasioned entirely by the wound, as being absolutely authenticated. In such cases as have lived for some time after the injury, later pheno-

mena may be produced by changes in the pericardium or endocardium.

The only instance of wound of the heart which has ever



FIG. 186.—Bullet wound through conus arteriosus.

been directly under my own observation was that of a young man who received a bullet at close quarters from a small revolver. The ball entered the chest in the third left intercostal space  $1\frac{1}{2}$  in. from mid-sternum. On examining the body

the pericardium was found distended with blood-clot, and the heart was firmly contracted in systole. The bullet had passed through the highest portion of the conus arteriosus, traversing it from front to back, and had also cut off the tip of the left auricular appendix. The appearances are seen in Fig. 186, which shows the heart in profile taken from the left side. The rod indicates the points of entrance and exit of the bullet, and shows the damage done to the auricular appendix.

## CHAPTER XV.

### COMPLEX SENSORY AND MOTOR AFFECTIONS.

A LARGE number of conditions essentially characterised by alterations in the nervous activities of the heart must be considered in this section. They are to be regarded not as diseases, but as symptoms, or groups of symptoms. Such disorders are frequently found to have a basis of structural alteration ; it is therefore inaccurate to speak of them always as functional diseases. In the same way many of them cannot be regarded entirely as neuroses, and the most satisfactory term for the group, therefore, is that which heads this chapter. The individual features have already been described in the chapters upon symptoms, and some of them have also been discussed in relation to certain cardiac lesions. It is convenient, however, to consider them from another point of view, gathering together those which form groups of symptoms, as they frequently do. In considering these disturbances, those only which concern the heart itself will be studied ; changes solely affecting the vessels will not be discussed, as they would lead to matters beyond the proper sphere of this work.

Many different arrangements of the subject have been made by various authors, and by Walshe, more especially, there has been much refinement in the subtle classification of dynamic diseases, as they are called by him. The simplest course is to divide the various affections into sensory and motor. These, however, are often associated together variously and capriciously, as Walshe says, and a classification, based upon such a fundamental plan, must only be adopted



with the reservation that any given affection, while no doubt tending more towards either the sensory or the motor type of disturbance, is usually found to implicate both.

### SENSORY AFFECTIONS.

The sensory changes have been previously described and analysed so completely that nothing more requires to be said about them as isolated phenomena, but there is a complex of symptoms forming one of the most characteristic affections of the circulation, of which a detailed description is necessary.

### ANGINA PECTORIS.

The group of symptoms known, from the date of their earliest scientific analysis, by the term *angina pectoris*, forms a variable picture, produced by morbid conditions scarcely, if at all, less diverse in character than the clinical appearances.

From internal evidence, it is by no means unlikely that the philosopher Seneca referred to the symptoms of *angina pectoris* in his own person, but the features of the affection under which he laboured, severe as they were, leave its exact character in doubt. "*Omnia corporis aut incommoda aut pericula per me transierunt: nullum mihi videtur molestius. Quid ni? aliud enim quidquid est, ægrotare est; Hoc est animam agere.—Ego vero et in ipsa suffocatione non desii cogitationibus laetis ac fortibus acquiescere.*" Such, in his own words, is the description of his sufferings. It is interesting to note his further remark that the attack came on suddenly and persisted about an hour.

Huchard states that Gaspard de Schomberg died probably from *angina*, and that his son also perished from heart failure, preceded by oppression and pain in the chest. The same authority cites the account by Lord Clarendon of his father's sudden death after severe agony in the left arm. A very clear description is given by Hoffmann of an undoubted case of this affection. The patient, a man, suffered from pain and oppression in the *præcordia*, radiating in all directions, and especially passing down the arms.

A graphic description of the symptoms of this disorder,

along with an account of the lesions, is due to Morgagni. The patient in this instance was a woman, who died in the year 1707, at the age of forty-two. The characteristic distribution of the pain, the fact of sudden death during a paroxysm, and the existence of aortic lesions are fully recorded.

French authors have put forward a claim for priority on behalf of Rougnon, who addressed a communication to Lorry, describing a case of pain in the chest followed by sudden death. Diligent search amongst the libraries in this country, and also in Paris, for assistance in which latter quest my warm thanks are due to Dr. Hahn, Librarian to the Faculty of Medicine in Paris, entirely failed to bring this letter to light; but, through the kindness of Dr. Merrill, Major and Surgeon, a type-written transcript has been furnished to me from the copy contained in the Library of the United States Army Medical Museum, now under his direction. As its contents are but indifferently known, it seems advisable to give a somewhat full analysis of them. The letter is dated 18th March 1768, and begins by referring to a short communication made by Rougnon to Lorry three weeks previously. The patient, whose case formed the subject of the letter, was fifty years old, and had for a long period served with distinction as Captain in the Dauphin Regiment of Cavalry, but he had retired some years before his illness from the army, and led a quiet life at Besançon. He had become fat, and had suffered from severe attacks of intermittent fever, which were followed by some jaundice, easily removed by diet; but for some years he had complained of difficulty of breathing on exercise. He attempted by a rigid diet to obviate a tendency which he feared might be asthmatic, yet the respiratory difficulty became more troublesome, so that he could not walk a hundred paces at all quickly without experiencing some feelings of suffocation. These, however, passed off after rest. About six weeks before his death the patient confided to Rougnon that during the breathless attacks he suffered from pain over the front of the chest; in the words of the letter: "*Il éprouvoit une gêne singulière sur toute la partie antérieure de la poitrine, en forme de plastron, & qu'il ne pouvoit faire une inspiration un peu profonde.*"

When the patient kept at rest and did not speak he scarcely experienced a trace of this embarrassment. The symptoms steadily increased in severity until the 23rd February, when the patient dined with some of his friends, and afterwards sat down to cards. The game lasted rather longer than he had intended, as he had an appointment which he was particularly anxious to keep. He left in a hurry, anxious to be present according to appointment at another gathering, and reached the house in a condition of great oppression; he leaned for a moment against the door in order to rest, and a servant, thinking him ill, offered his services, which, however, were declined by the patient, who, feeling somewhat relieved, thanked him, and went upstairs. Walking somewhat precipitately, he reached the apartment in a state of oppression, sat down amongst his friends, and was at once observed to be dying. He was forthwith carried out and found to be dead.

On the following day a post-mortem examination was performed. The brain was perfectly healthy. The walls of the chest presented an extraordinary hardness of the costal cartilages, which were ossified, more especially in the upper part of the chest. On removing them and the sternum, it was found that the pericardium was the seat of a large accumulation of fat. The lungs were healthy, with the exception of some slight adhesions. The heart was larger than usual, apparently on account of dilatation of the right ventricle, the walls of which were considerably attenuated, while the cavity contained a large quantity of blood, which had scarcely coagulated. The right auricle was greatly dilated, as was also the vena cava, the diameter of which was about 2 in. near the heart. Both of them contained fluid blood. The cardiac and coronary veins were much distended, and in a varicose condition. The left ventricle and auricle, as well as the pulmonary veins and aorta, were of the usual size, and entirely empty.

Having given these details, Rougnon then enters upon a long and diffuse account of the circulation of the blood, in order to serve as the basis of an argument explanatory of the death of the patient. It is not possible to review the assertions and arguments which Rougnon offers, but a brief abstract of his views may be permitted. The ossification of

the cartilages interfered with inspiration, prevented the passage of the blood through the lungs, and thus produced stasis in the right side of the heart, causing dilatation of the right ventricle and auricle, as well as of the vena cava and coronary veins. He further adds that the accumulation of fat around the heart must have still further interfered with the respiration. He emphasises the fact that on the evening of his death the patient had been worried by the fear of being late for his appointment, and that, with the barometer standing at 27 in. and 2 lines of mercury, he had hurried in order to keep his engagement; the consequence of which was that the feeble contractions of the heart were incapable of driving the blood through the lungs, so that, ceasing to receive blood from the pulmonary veins, the left ventricle failed to contract. The letter ends up with some suggestions as to the diagnosis of ossification of the costal cartilages, and the means by which such ossification may be prevented or removed. There can be but little doubt that the case described by Rougnon was one of cardiac pain, but the description entirely lacks the special features described so fully by the great writer who must now be mentioned.

These are the only references which can in any way bear upon this affection, until the epoch-making researches of Heberden called the attention of the entire world of medicine to the complex of symptoms named by him *Angina Pectoris*. In his first communication, read 21st July 1768, he published investigations, spread over more than twenty years, dealing with observations on about twenty patients, which contained a full description of most of the symptoms now known to us.

A letter to Heberden from an anonymous writer, dated 16th April 1772, and read by the former 17th November 1772, contained a most graphic account of the subjective symptoms as detailed by a skilled observer, and in it there is recorded an experience of sensations which "seem to indicate a sudden death," then mentioned for the first time, unless the phrase used by Seneca, "*Hoc est animam agere*," should perchance refer to it. Heberden described the appearances found by John Hunter after death in this patient, there being no cardiac change except a slight ossification of the



aorta. In his commentary on the case, Heberden mentions that he cannot remember to have heard previously of the sensation of an apparent suspension of life, or, as the anonymous writer puts it, "an universal pause within me of the operations of nature for perhaps three or four seconds."

Wall, in a letter to Heberden, dated 30th May 1772, gave a good description of a case of this affection, in which after death there was found a great change in the aortic cusps. Haygarth also in the following year read a case, but evidently misapprehended the post-mortem appearances. Fothergill published two papers dealing with angina pectoris, in the second of which he described the anatomical results found in a patient examined by John Hunter in 1776. In this case ossification of the coronary arteries was observed.

The discovery of a change in the coronary arteries, associated with præcordial pain, seems to have been made, before the date mentioned above, by Jenner. Jenner addressed a letter to Heberden in which he described changes in the coronary arteries. His observations were discussed by a private medical society in Gloucestershire, and were afterwards embodied in a communication addressed to Parry, who read a paper on the subject in July 1788 before the society; this paper was published in an extended form by Parry eleven years afterwards, and in it the author strongly advocated Jenner's views. It is quite clear that the reason for Jenner's reticence, in regard to the views to which he had been led, was solicitous consideration for the feelings of Hunter, who had begun to manifest some anginous symptoms.

The anatomical fact of ossification of the coronary arteries was observed by Bellini and many of his successors. The connection between the change and anginous symptoms was left for the sagacious acumen of Jenner to discover.

Black was the next observer who brought forward a coronary change in angina pectoris, and it is most interesting to note that the patient, in whom the lesion in the coronary vessels was afterwards seen, complained of an intolerable sense of anguish at the heart, resembling that which is felt by a person exhausted and ready to faint from running.

In his *Commentaries*, published the year after his death, Heberden mentions that he had seen not less than one hundred cases of angina pectoris, of which there occurred three in women, and one in a boy twelve years old; all the others were in men of above or nearly fifty. He somewhat amplifies his clinical description of the affection, but adds nothing of importance to the facts of morbid anatomy. It is impossible to ignore the circumstance that he is silent regarding the state of the coronary arteries. The only direct reference to structural change is contained in the sentence: "Inciso cadavere hominis, qui hoc morbo subito perierat, expertissimus anatomicus nullum vitium deprehendere potuit in corde, aut in valvulis, aut in arteriis, venisve vicinis, præter exigua rudimenta ossea in aorta."

Until the appearance of the work of Latham, but little of interest was added to the knowledge of the affection, and the chief symptom brought into strong relief by this accurate observer is the sense of impending death during a paroxysm. Such are the earliest records of original investigation upon this subject. An enormous literature upon the subject has since then accumulated. Almost every writer upon diseases of the heart has given utterance to his own particular views on this subject, many of which must be referred to in the sequel; it is, however, obviously impossible to dwell at length upon the purely historical aspect of the observations and opinions which have been published in regard to angina pectoris. The more recent writers, from whom most luminous opinions may be gathered on this subject, are Latham, Gairdner, Huchard, and Osler.

ETIOLOGY.—The causes which lead to the development of angina pectoris may be said to be those which produce the conditions underlying it. These various factors must be regarded in the light of predisposing causes.

Heredity exerts a very prominent influence in the evolution of the complex of symptoms. It is a fact universally recognised that certain families are more particularly prone to degenerative processes affecting the arterial system and the heart—processes which must be allowed the most prominent place in the production of the affection. Many instances are

now on record of the affection following a family through two or more generations, of which the most important as well as the most interesting example is that of the three generations of Arnolds, fully discussed from different historical points of view by Latham, Gairdner, and Osler.

With regard to sex, the affection is without a doubt much more frequent in men than in women. It is easy to account for this difference in liability. The male sex, by greater exposure to the manifold varieties of physical and mental stress, is more prone to the degenerative changes upon which most cases are grafted, while at the same time more liable to the excitement likely to act as determining causes of attacks.

Age exercises a considerable amount of influence in the development of the affection. It is relatively rare before middle life, and is found more particularly in those who are just passing from middle into elderly life. It has, however, been seen in much younger periods, although in these it is admittedly rare. A case, notwithstanding, is mentioned by Balfour in which the affection was present at the age of twenty-four years, which must be placed alongside of the fact that Wild determined the existence of coronary sclerosis in sudden death at the age of twelve.

Some occupations render those who follow them more particularly liable to attacks of angina pectoris. Long-continued physical exertion of severe character has already been considered as a most important factor in the production of arterial and cardiac sclerosis, and there cannot be a doubt that many of those whose avocations lead them to physical over-stress are liable to angina pectoris. In this place it is impossible to pass by the statements of some authors without comment. It has frequently been asserted that angina pectoris is far from common amongst hospital patients. This, however, cannot be accepted as an accurate statement, for in districts where hard mechanical occupation is the rule, angina pectoris is far from uncommon amongst working people.

Long-continued mental efforts appear to be attendant upon, if not productive of, the lesions so often underlying angina pectoris; it is probable that the reason for this is that such persistent intellectual labours exert their influence by leading

to continuous high arterial pressure. Reference may be made in this connection to an important paper by Clifford Allbutt on the connection of mental exertion and renal cirrhosis, as containing important observations in this direction.

Climate has probably something to do with the development of the lesions leading to angina pectoris. The affection is undoubtedly more common in northern regions, but it is probably less to the influence of cold than to the habits whither it tends that we must look for the production of these lesions.

Some general and constitutional disturbances are observed as predisposing causes of angina pectoris. Amongst these gout must certainly be allowed the most important place. The faulty metabolism constituting this condition leads to the lesions which so often predispose to it, as well as to some of the disturbances apt to precipitate an attack. Diabetes must also be mentioned in this connection.

Specific infection in the tertiary stage is an undeniable cause, operating through aortic or coronary endarteritis. It is probable that in most instances the infective process is associated with the abuse of alcohol. The occurrence of cardiac pain in ataxy shows the specific process acting at once on the circulatory and nervous systems.

Some of the acute infectious diseases give rise to angina pectoris; chief among these is influenza, but enteric fever and analogous diseases are also effective in this way. In a manner quite analogous, toxic chemical substances, such as tea, coffee, alcohol, and tobacco, lead to the affection.

The exciting causes which induce attacks are for the most part influences capable of inducing conditions of stress. These are sometimes purely mechanical, such as are often seen in connection with alimentary disturbances. Dilatation of the stomach or distension of the intestines may bring on an attack, while an unfavourable attitude unconsciously assumed during sleep is enough to precipitate an access. Physical exertion, especially after meals, is the most frequent determining agent. Mental efforts also induce attacks, but disturbing emotions, whether of joy, grief, or anger, are much more powerful. The influence of external agencies, such as cold and damp, must not



be overlooked, especially in the production of one distinct variety. It seems also probable that certain disturbances of the system may reflexly precipitate an onset. Some toxic substances, lastly, have an exciting as well as a predisposing tendency. Tobacco is the chief agent of this group, but tea, coffee, and alcohol also claim their victims.

MORBID ANATOMY.—There are, it need hardly be remarked, no special lesions belonging to angina pectoris, but some structural alterations are so commonly associated with the affection that a causal nexus may fairly be allowed. The lesions are exceedingly numerous, but may be classified under a few heads.

The myocardium is frequently the seat of chronic interstitial myocarditis, often associated with arterial sclerosis, which may be general, or may affect the coronary vessels in particular. So common is the connection between anginous seizures and interstitial myocarditis in its chronic form, that surprise is usually expressed when a fibroid change is discovered at the post-mortem examination of any patient who has been free from anginous seizures during life. It must, however, be admitted that a very large number of cases of chronic myocarditis have little or no cardiac uneasiness. Such a case has already been described (Case 53, p. 690). Fatty degeneration of the heart, resulting from fatty accumulation and infiltration, is not rarely accompanied by cardiac pain, amounting really to angina. This was observed by Jenner, as mentioned by Parry. This association has been observed more particularly by Douglas Powell, and it is illustrated by one of the cases described subsequently. Angina is found in association with gummata, and their attendant fibroid changes in the myocardium, but, seeing that such specific developments are comparatively rare, they cannot be regarded as common lesions in the affection.

Many lesions of the endocardium have been described, but it is impossible to regard any of these as being connected directly with the development of angina pectoris. As these lesions, however, are frequently produced by degenerative changes, there need be no wonder that some of them are found in association with cardiac pain. This is

more especially the case as regards the aortic orifice and its cusps. It has already been sufficiently insisted upon, in the chapter dealing with aortic lesions, that anginous paroxysms are common, and the obvious explanation of this is that in the degenerative affections of the aortic orifice there is apt to be some interference with the blood supply to the coronary arteries. This may occur in two different ways, as will be shortly pointed out. Mitral lesions, as well as organic valvular affections of the right side of the heart, are seldom accompanied by cardiac uneasiness, deserving the title of angina pectoris. The lesions of these orifices occur most commonly in early life, and arise from endocarditis. There is therefore less tendency in them to produce nutritive disturbances of the myocardium.

The arterial system exercises the most powerful influence in the evolution of angina pectoris. The widespread degeneration known as arterio-sclerosis, or endarteritis deformans, is more especially found. There is also, in a smaller number of instances, well-marked endarteritis obliterans, but instances presenting this lesion are almost always associated with the development of gummata in the myocardium.

Besides alterations of the aortic orifice, there are sometimes changes in the ascending aorta. Acute or chronic aortitis, fusiform dilatation, and sacculated aneurysm are all found.

The essential importance of all these changes lies in their influence upon the nutrition of the heart. Structural alterations in the aortic walls are very prone to cause more or less occlusion of the mouths of the coronary arteries, while sclerotic changes in the arterial system are more frequent in the vessels of the heart than elsewhere, probably from the great oscillations of pressure to which they are subjected. Interference with the mouths, or sclerosis of the trunks, of the coronary arteries lessen their nutritive capabilities, while the latter process leads to occlusion by thrombosis. Senac has, nevertheless, described a case of coronary ossification without any pain, and the same has often since been recorded. Such an instance has been seen in Case 53, already referred to. Whether embolism of the coronary arteries gives rise to angina pectoris is doubtful.

Lesions of the nervous system have been found in the affection. Neuritis of the cardiac plexus alone, or of the plexus and the phrenic nerve, has been described by Lancereaux and Peter, and has already been considered in a former chapter.

Such are the most important of the lesions which have been described in cases of angina pectoris.

SYMPTOMS.—In angina pectoris the objective are dwarfed by the subjective features. It follows from this consideration, as Gairdner has mentioned in his masterly analysis of the affection, that it is difficult to distinguish between the essential facts and the speculative conceptions connected with the disorder. The former are so largely beyond the range of exact observation on account of their subjective character as to be in a great degree elusive, and they are further apt to be interwoven with, or overshadowed by, ideas of a more or less unsubstantial nature, according to the individual tendencies of the patient in any given case.

Pain is the great central fact in angina pectoris; the other symptoms group themselves as it were around it. It has already been shown that cardiac pain varies infinitely in degree, ranging from slight uneasiness to the most overwhelming anguish.

In the analysis of symptoms connected with the nervous system, belief has been expressed that there is no real distinction between different varieties of pain associated with cardiac disturbance, save that of degree. There are many considerations which prove the correctness of such a view. Slight degrees of cardiac pain may be found associated with profound structural alterations such as commonly underlie severe angina. On the other hand, painful sensations of the most serious character and typical distribution are to be found along with slight structural alterations and transitory toxic processes. In the same patient there is not infrequently an alternation of attacks of greater and lesser severity. It must further be kept in view that the less serious varieties of pain are connected with those of the gravest character by intermediate varieties of every degree, so as to form a chain whose links are unbroken.

The pain experienced in pronounced angina pectoris is intense in degree—so overwhelming, indeed, as to merit the term anguish often applied to it. In certain cases it is developed with startling suddenness, but in other instances it attains its maximum gradually. An essential character of the affection is its paroxysmal nature. Attacks are frequently induced by exertion, physical or mental, and some of the most classical cases on record have come to a dramatic termination from such causes.

The distribution of the pain, although presenting many variations, is nevertheless remarkable in that it is almost invariably confined to certain recognised areas. It is most commonly felt deeply seated behind the lower half of the sternum, but, less frequently, patients also describe it as in the upper sternal region. Occasionally it is not experienced in the sternal region at all, but is situated in one or other of the sides of the chest, usually the left, the reason for which has been already stated. The extent of the area over which the pain is felt often varies with different paroxysms, not only in extent, but also in position.

A special feature of the pain is its tendency to radiate in certain definite directions—to follow, as may be said, great trunk lines. It most commonly extends upwards to the left shoulder, or to both shoulders—very rarely to the right alone, and almost as frequently reaches the elbow, or even the fingertips. It is sometimes found near the angle of the scapula, on one or both sides, and now and then a patient will describe it as occupying a wide area on the back of the chest. Somewhat less commonly the pain is felt over the neck and occiput. The painful sensations may, however, be found to extend far more widely than has already been mentioned, and are sometimes even experienced in the lower parts of the trunk and in the lower limbs. Examples of the distribution are shown in the figures which accompany some of the cases narrated below.

Very frequently, so often in fact as almost to be the rule, the pain is attended by a sense of constriction within the chest. This feeling, variously phrased by different patients according as they are able to analyse their own sensations, is essentially one of tightness or compression.



A numb feeling in some part, more especially of the arm, is common in angina pectoris, but it presents itself in a variable manner. It is in some cases a constant symptom, retaining a fixed position even during long periods. In other instances patients only experience the sensation of numbness during a paroxysm.

Other sensory symptoms may come under the notice of the physician, more rarely, however, than those just mentioned. Of such subjective phenomena the less infrequent are probably tingling, creeping, and similar feelings. Subjective sensations connected with the special senses are by no means uncommon. Disturbances of sight and hearing, sometimes accompanied by giddiness and vertigo, may be determined.

Perhaps the most frequent description given of this symptom by those who have suffered from angina pectoris is that of a want of breath, and in truth the respiration is frequently irregular, unequal, and shallow. There is, however, no difficulty in breathing. If the patient makes the effort, he is able without difficulty to fill the chest with air.

But over and above all these subjective manifestations of profound disturbance there is yet another, which in its overwhelming nature and tremendous import overshadows even the unbearable anguish of the pain. This symptom is the sense of impending death. In some degree at least, this feeling is always present in every paroxysm of angina pectoris. It seems for the most part to be a symptom associated with the pain, yet there are not wanting certain facts which go far to disprove that it is in any sense the result of it. It is undoubted, for example, that, in the fatal seizures by which life is so abruptly ended, patients, although they may have suffered the most extreme anguish from time to time during previous attacks, are suddenly visited by a sense of imminent death, without any painful sensation, and pass away without a struggle. This is the "angina sine dolore" of Gairdner. Even in the earliest notice of this sensation, contained in the letter to Heberden from his unknown correspondent, it is said that the foreboding of imminent death is not concomitant with the pain.

The sensation of approaching dissolution is probably

caused by some inhibitory influence. It belongs, as has been seen, to the same class of depressing sensations as are found in many visceral affections accompanied by severe pain, some of which lead, if unrelieved, to syncope, or even asystole. And if fainting or death may occur in such an affection as gallstone, how much more likely is it to take place in an anginous paroxysm! For, with a nervous mechanism including depressor fibres, there is a special provision for interferences with the functions of the heart, attended by the most depressing subjective manifestations. Those able to describe and analyse sensations, who have passed through the ordeal of angina pectoris, speak of another feeling which they have experienced along with the sense of imminent death—that of sinking. And the sensation of sinking is in some cases accompanied by a feeling as if the heart were stopping.

It is more than probable that in all cases where the other symptoms of angina pectoris are attended by these characteristic sensations of sinking, of stoppage of the heart, and of the near presence of death, there is a direct stimulation of depressor fibres leading to inhibition of the cardiac pulsation.

There cannot be any question that in almost every grave case of angina pectoris there is a tendency to syncope, that the heart, in short, is struggling to meet or overcome an obstruction to which it is inadequate, and the old synonym, syncope anginosa, introduced by Parry is singularly applicable to many cases of the disease.

The countenance, even in the less severe forms of angina pectoris, frequently betrays uneasiness or anxiety; in the more serious attacks it usually betokens unutterable anguish and fearful anticipation. The face is usually pale, but this is a rule liable to many exceptions, and it is by no means uncommon to observe a general or local flush, while extreme lividity may be present. The surface of the skin is in a large proportion of cases bedewed with perspiration during the climax of the paroxysm.

The posture assumed by a patient during an attack is striking. If a paroxysm should occur while the patient is on his feet, he will usually be found leaning against, or clinging to, some object until the symptoms have abated; if an attack

should arise while he is in bed, he will be seen sitting up and leaning forward so as to rest his arms on his knees.

The pulse has been the subject of considerable discussion, and the explanation of the discrepancy of views expressed in regard to it can only be that the pulse presents variable appearances. Since the earliest observations of Lauder Brunton, it has been well known that the pulse pressure is often high, and it has been assumed that this is an invariable accompaniment of the affection. This, however, cannot be admitted. Angina pectoris is sometimes unattended by any increased arterial pressure. Morison has, more particularly, directed attention to this point. The pulse varies considerably as to rate and regularity. In some cases its rate is moderate, and the rhythm may be perfectly regular, while at other times very opposite conditions prevail. The pulsations are sometimes almost imperceptible; it was so in the case of John Hunter, and the same condition has often been observed since.

On examination of the neck and præcordia, there may be slight or grave departures from physiological conditions. There may, for instance, be pulsation of the veins of the neck, or very extensive pulsations of the carotid arteries. The præcordia may show a wide area of impulse—forcible and sustained if there is much hypertrophy; weak and fugitive if dilatation be in the ascendant. The area of dulness may be very considerably increased either to right or left, or both, and on auscultation, murmurs denoting different valvular lesions may be determined; in short, extremely variable conditions of the heart may be present. In the majority of instances, however, physical signs indicating some affection of the left ventricle, aorta, or aortic cusps may be determined by the physical signs which are present. These are the most frequent morbid conditions ascertained on clinical examination.

The alimentary system shows symptoms of disturbance in flatulence, hiccough, retching, or vomiting, which may possibly, as von Dusch long ago suggested, be due to implication of the nervous system.

Respiratory symptoms are common. During an attack the respirations are almost invariably shallow, as if the patient feared to aggravate the pain by breathing. If a deep breath

is attempted, it is found to be easily drawn, and is sometimes described as bringing positive relief. Continuous yawning is not at all infrequent, and it must, like hiccough, be produced by some reflex nervous mechanism.

Disturbance of the urinary functions is only shown by frequency of micturition during the period succeeding an attack, along with an increase in the amount, and a low specific gravity of the secretion.

The nervous symptoms which are commonly associated with, but overshadowed by, the principal feature have been sufficiently discussed.

Over the regions in which the subjective sensation of pain is experienced there is always hyperæsthesia, when tested in the manner previously indicated. As in the case of subjective pain, this exalted sensibility is most commonly found over the distribution of the lower cervical and upper dorsal nerves. It is certainly found more frequently over the area supplied by the internal anterior thoracic nerve, from the eighth cervical and first dorsal roots, than elsewhere; but it is very commonly present as far down as the fifth or sixth dorsal nerve.

Although cases of angina pectoris present an almost perfect series of symptoms with different degrees of intensity, it is yet possible to select certain of these, and regard them as types. That which has been described may be regarded as the most definite variety, from which the series passes down by imperceptible gradations to others of much less serious import.

Toxic conditions may, and often do, give rise to angina pectoris. The chief agent producing the complex of painful symptoms is tobacco, but alcohol gives rise also to more or less pronounced features. My own experience has invariably been that in these cases there is cardiac dilatation. Tea and coffee are blamed for similar effects, but such have never come under my notice. There are, doubtless, many uncomfortable feelings produced by the abuse of these substances, but most of them are of gastric origin. In most toxic cases there can be no doubt that the effects are due to an intimate chemical union of the poison and the protoplasm of the nervous and muscular substances—one or more atoms of some chemical body of the



one being replaced by a corresponding amount of the other. What may be the unstable chemical element, and what the particular affinity, must needs be, with our present knowledge, mere speculation.

Under the head of neurotic angina pectoris, Osler includes several varieties of what were termed by Walshe pseudo-angina pectoris. The various varieties which may be included under this head seem to me to be four in number.

Reflex angina pectoris, first observed by Landois, may be produced by many different kinds of irritation, but gastro-enteric disorders, pulmonic affections, and nervous disturbances form the most frequent causes. In many of the cases usually placed in this group there is without doubt some underlying structural alteration, and the reflex factor can in such instances only be regarded as the exciting cause. With all due allowance for cases of this description, there are, nevertheless, occasional examples of what can only be regarded as reflex angina pectoris.

The vasomotor type was first described by Nothnagel. It is characterised by sudden onset of coldness or lividity of the extremities, often associated with profound perspiration, and attended by severe cardiac pain, along with palpitation and feelings of faintness. The symptoms are most commonly seen in cold weather, but may set in while the patient is warm in bed. These appearances are not uncommon in patients who undoubtedly present evidence of the structural alterations found in cases of classical angina pectoris. They occur, nevertheless, in the absence of all such structural alterations, and it seems probable that Nothnagel is perfectly correct in regarding their origin as due to vaso-constrictor influences acting through the arterial system upon the heart. It is easy to draw a parallel between instances of this kind and Raynaud's Disease.

Neurasthenic angina pectoris occurs in patients of neurotic predisposition, who have passed through periods of long-continued overwork. In this variety of the affection there may be very severe, even agonising, pain in the præcordial region, extending to one or both shoulders, and thence down the arms as far as the finger-tips. Such attacks are frequently accom-

panied by feelings of fluttering or faintness, along with sensations of cold over some part of the surface, which may appear either pale or livid, and is often bedewed with perspiration.

The hysterical type is found as one of the protean manifestations of that singular affection, and is usually accompanied by other symptoms, as well as by the characteristic background upon which the appearances are, as it were, depicted. In this type, severe pain is sometimes complained of in the præcordial region, with a tendency to radiate to the shoulders and arms, and, along with this, various vasomotor appearances may be in evidence on the surface. One or two points of interest may be noted in such conditions. The patient has very commonly some points of hyperæsthesia—the hysterogenic spots—as well as anæsthetic areas. It is further to be remarked that the area to which the subjective sensation is referred is very often objectively hyperæsthetic to an extreme degree. It may, indeed, be regarded as a hysterogenic spot, and, as will be seen in one interesting case recorded in the sequel, pressure on such a point may produce curious nervous symptoms.

It has been remarked that all varieties and degrees of cardiac pain pass by imperceptible gradations into each other. They cannot, therefore, simply by the character of the pain be differentiated. It is not difficult, however, in almost every instance to determine to which category any given case belongs. This subject will be considered under the head of diagnosis. Before leaving this aspect of the question, it may be well to refer to the use of the term *pseudo-angina pectoris*. The great authority of Walshe has led to the common employment of this phrase, and it is to be regretted that such has been the case. Strictly speaking, there cannot be any condition meriting the name *false angina pectoris*. The term is undoubtedly objectionable, and many modern authors, such as Balfour and Morison, have frankly said so. It is unfortunate that in his interesting lectures upon this subject Osler has employed the term. The various kinds of cardiac pain which have been described are all equally real, whether they be linked with grave organic disease or not, just as a facial neuralgia may be equally severe in one patient who has no discoverable lesion,

and in another in whom the pain depends upon pressure on a nerve trunk. The pain of angina pectoris may doubtless be simulated by imposture, just as trigeminal or any other neuralgia may be affected from a desire for effect, a longing for sympathy, or a craving for anodynes. This, however, is beside the mark, and because one malingerer may mimic the symptoms of facial neuralgia, we do not regard all neuralgia as false. So it is in the case of angina.

The course of angina pectoris necessarily depends, in the first place, upon the variety of pain which is present, and in this lies the great distinction between the different types. In that which is associated with aortic, coronary, or myocardial changes, there is great danger of a sudden termination of the affection. So much is this the case, that, as occasionally happens, a patient may be cut off in a first attack of pain. It occasionally happens that death occurs without any indubitable evidence that there has been any pain; the sudden termination of life in such cases is, as it has been termed by Gairdner, a "fragment" of observation. In many instances, however, as in the classical instance of the great headmaster of Rugby, described by Latham, death occurs in the first paroxysm of angina pectoris.

Much more commonly there are recurrent attacks spread over some months or years, in which the termination almost invariably takes place from sudden asystole. This termination is sometimes attended by the anguish of angina pectoris, but at other times the last phase of life is marked by a period of quietude, in so far as acute pain is concerned, yet there is much suffering on account of the syncopal attacks, which are apt to be accompanied by breathlessness, giddiness, and faintness. This is the phase termed the anginous state by Huchard, and it may be regarded as a form of the angina sine dolore of Gairdner. Those fond of learned phraseology might well give to the condition the name *status anginosus*.

A matter of great interest is the undoubted tendency of the paroxysms to become less frequent and less severe when dilatation of the heart supervenes in cases which have not previously been characterised by this condition. Musser has done good service by emphasising this fact. In most instances this alteration in

the condition of the heart leads to angina sine dolore, and is followed by death from asystole.

The symptoms are certainly very much modified by the habits of the patient. In those who guard against any serious physical stress, and who attempt as far as possible to maintain calmness of mind, there is much less likelihood of anginous seizures. When there is considerable physical and mental exertion, there is much more liability to violent attacks. The condition of the digestive viscera is also of the greatest importance, and if there be any carelessness as regards the primary digestion, more particularly if distension of the hollow viscera is allowed, there is apt to be, as a consequence, some impairment of the heart which may excite an access of the malady.

In the toxic varieties of angina pectoris the progress of the affection is, as a rule, satisfactory. The effects of the various poisons generally lead to their discontinuance, and, after a longer or shorter period, the symptoms disappear. In the tobacco heart, accompanied by cardiac pain, the symptoms are sometimes extremely persistent, no doubt on account of chemical union between them and the muscular or nervous structures. In reflex, vasomotor, hysterical, and neurasthenic angina pectoris, the great feature is that with longer periods of intermission any time of storm and stress is apt to bring about a recurrence of the paroxysms, which have fortunately but little tendency to shorten life.

The duration of an individual attack varies considerably. Sometimes it may occupy a few seconds, more commonly it is continued for several minutes, or may even be prolonged for two or three hours. It is a rather interesting point, to which Huchard has recently called attention more particularly, that the longest attacks are those which occur spontaneously, more especially during the night. Very commonly the anginous seizure passes away as soon as the exciting cause has been removed. If it occurs in the course of some physical exertion, it ceases at once when this is discontinued. If it is produced by any mental excitement, it passes away when a calmer psychical state is attained. In distension of the stomach it terminates abruptly on the relief of that condition.



The most important opinions which have been expressed as to the nature of the pain in angina pectoris have been duly stated in the analysis of symptoms. It is, therefore, quite needless to enter fully into them again; it is only necessary to state that the origin of cardiac pain is undoubtedly variable, and that its explanation in different instances is certainly manifold. Imperfect nutrition of the sensory endings of the cardiac nerves, whether occurring in the course of general anæmia or local ischæmia; toxic influences, whether by means of such chemical poisons as tobacco and alcohol, or such organic agencies as bacteria and their products; direct irritation, as in the hyperæmia and exudation of pericarditis, or in the structural changes found in myocarditis and myocardial degenerations, give rise to conditions capable of arousing sensory disturbances. In some instances there can be no doubt that afferent impulses are directly transmitted to the nerve centres, but it is probable that in others a condition of exaltation is produced, in which any additional strain, such as the struggle of the heart to overcome a resistance to which it is not adequate, may give rise to impulses which travel in a centripetal direction. Such a conception as this brings together the most important opinions of writers upon this difficult subject. All of those which have been mentioned are of value, but many of them are characterised by narrowness of view, only one aspect of the subject having been present in the mind of the writer.

DIAGNOSIS.—The questions requiring answer resolve themselves into two—whether painful symptoms are really connected with the heart; and, if this be the case, to which category they belong. There is, in general, but little difficulty in determining whether painful sensations are associated with the heart or not. The accompaniments of the pain as regards the state of the pulse and the heart, and the characteristic seat and distribution of the sensation, as well as its special mode of onset, are usually so distinct as to leave no room for doubt.

The recognition of the grave forms of the affection associated with myocardial, coronary, and aortic lesions is usually free from much difficulty. The presence of the physical signs

of cardiac and arterial degenerations forms the most reliable guide in this matter, while the age of the patient will also be found useful. The onset of the painful sensations, during exertion for instance, has some importance in diagnosis.

The forms of angina belonging to the toxic and neurotic groups may be distinguished from that attendant upon organic lesions of the arteries and heart by the absence of any evidence of such changes. When this has been determined, the diagnosis of the particular variety rests in great part upon the causes which may be found. The statements of patients, therefore, must in great part be depended upon, especially as regards the toxic forms. No doubt the effects of the poisons may be detected by attendant symptoms, even when denied by patients. This is not, however, invariably the case, and some doubt may exist as to the true character of the pain. Nevertheless, alcoholism may be recognised by the behaviour of the tongue and skeletal muscles in most instances, while nicotism is often revealed by the irritation of the fauces.

Reflex angina requires careful search after its causes, while in the vasomotor, neurasthenic, and hysterical forms there is sufficient evidence in the other symptoms which are present to warrant a diagnosis.

PROGNOSIS.—The apparent severity of attacks may be similar, yet the absolute danger vastly different; the attempt to forecast the future is in consequence beset by difficulties of no ordinary kind.

In angina pectoris having for its basis grave structural changes in the heart the prognosis must always be serious. When associated with general arterial sclerosis, which may be presumed to have invaded the coronary arteries, and through them to have produced chronic interstitial changes in the heart muscle, evidenced by symptoms of cardiac failure consecutive to hypertrophy, the outlook is certainly grave indeed. If, in addition to this group of appearances, there is also incompetence of the aortic cusps, the element of danger is greatly increased and the prognosis much more gloomy. The same consideration arises if, along with the arterial and myocardial lesions, there is evidence of chronic renal changes.

Those forms of angina pectoris which occur in the course

of fatty accumulation and encroachment are by no means so serious, and patients often live for many years with more or less frequent seizures according to external circumstances. The prognosis in such cases is therefore much more hopeful than in the sclerotic type.

Toxic angina pectoris is seldom of grave prognostic import, and on the removal of the prime factor the symptoms usually disappear for good.

In the neurotic forms of the affection the outlook is, as a general rule, perfectly satisfactory as regards life, but there is a great tendency to recurrence.

TREATMENT.—The means of relief in sensory disturbances connected with the heart will be sought in directions according to the views held with regard to their causation. Yet it must be admitted that the treatment of cardiac pain rests, for the most part, with a few noteworthy exceptions, on the results of empiricism.

The treatment of the classical type of angina pectoris necessarily resolves itself into the management of the general health, and the immediate relief of pain when present.

The general treatment of any patient in whom pain is a prominent symptom must be an attempt, through restoring the general health, and more especially improving the circulatory and nervous systems, to procure prophylactic influences as regards pain. The air and light, the regulation of rest and exercise, the arrangement of food and drink, and the employment of such remedies as are useful, require due attention.

In the treatment of cardiac pain it is necessary that the whole digestive apparatus should be in good working order. Not merely does a sluggish digestion cause mechanical interference with the circulation in many ways, but chemically through the blood it interferes with the metabolic functions of the heart. The respiratory organs must in the same way be watched, and if there be any mechanical interference, by means of bronchial affections, with the proper aeration of the blood, it must be rectified. The nervous system must, further, be calmed if there be any excitability or irritability.

When pain is obviously associated with failure of the

energy of the heart, cardiac tonics will be required, and, according to circumstances, such a drug as digitalis or strychnine or arsenic may have to be employed. Many of the conditions of failing heart which underlie angina pectoris receive much benefit from the use of baths, massage, and exercises. This has already been fully described.

Amongst the remedies useful from the prophylactic point of view, the iodides are pre-eminent. One of the most remarkable effects produced by the iodides is the relief of painful affections. The most famous of these drugs is iodide of potassium. This drug, first introduced by Graves in the treatment of painful diseases of the fibrous tissues and nervous system, was observed by Craig to have produced perfect relief from pain in a case of aneurysm, as he stated in a private communication to Balfour. The use of iodide of potassium appears shortly afterwards to have been almost simultaneously investigated in Europe and Asia by different observers. Bouillaud in Paris, and Chuckerbutty of Calcutta, found that great relief was obtained by the use of iodide in cases of aneurysm. Roberts of Manchester, somewhat later, employed this remedy on a considerable scale, but it is to Balfour of Edinburgh that we owe the extended employment of the drug. Unfortunately, we do not yet understand the mode of action of the iodides. It is, however, well known that they have some influence in reducing the pressure and diminishing the frequency of the pulse. The amount of reduction of blood pressure, however, is extremely small, and the diminution in the rate of the heart is inappreciable with ordinary doses of the drug. It has been shown by Sée and Lapique that the first effect of iodide of potassium in a small dose is to increase the blood pressure and to diminish the frequency of the pulse, and that, if the dose is increased, the blood pressure falls, while the rate of pulsation rises. We know, further, that in addition to these effects upon the circulation, the iodides have considerable effect in removing morbid products and restoring healthy structure. When all these effects have been taken into account, we are still left uncertain as to the precise mode of action of the iodides in relieving pain, but its removal is one of the most



constant and most useful effects produced by this series of drugs.

Amongst the iodides, iodide of potassium is that which has been most constantly employed, and which is probably in most respects the best. Sée has pointed out that, in comparing the iodides, the salts of calcium, sodium, potassium, and strontium stand in the order in which they have been mentioned with regard to the amount of iodine which they respectively contain; that of calcium containing the most, and that of strontium the least, while the salts of sodium and potassium occupy an intermediate position. The difference between the different members of the series is, however, insignificant, and the facts cannot be allowed much weight.

In addition to the iodides of the alkalies or alkaline earths, hydriodic acid may be usefully employed in the form of a syrup. Another way in which iodine may be continuously administered is by means of iodine wine, a preparation which has given excellent results. The use of arsenic along with the iodides often appears to aid them in effecting improvement. The dose of any iodide should not exceed 10 grains, with which 5 minims of the arsenical solution may be combined. Hydriodic acid is employed in minim doses, and of the syrup the dose is 1 drachm.

Since the observations of Lauder Brunton, the nitrites have been most extensively used in the treatment of every form of cardiac pain. Nitrite of amyl, applied by Lauder Brunton in the first instance, is, from its nature, more especially applicable to the relief of paroxysms of pain when present, and in this connection it will be mentioned immediately. Every member of the group of nitrites is endowed with the special action of dilatation of the arterioles, and the choice of any of them must be based upon their relative value in this respect, and their relative freedom from danger. Spirit of nitrous ether, long employed in ignorance of its vaso-dilator effects for many purposes; nitro-glycerin, suggested by Murrell; and nitrite of sodium, introduced by Hay, produce similar effects, in different degrees. There are, however, immense differences in regard

to the dangers attendant upon these drugs. While nitrous ether is free from all danger, and nitro-glycerin has few disagreeable attendants, sodium nitrite is often followed by alarming symptoms, and cannot be regarded as a drug to be indiscriminately prescribed. Quite recently erythrol tetranitrate has been introduced by Bradbury. From the few observations which have been allowed me since the drug was brought forward, no doubt has been left in my mind as to its value. Although somewhat less rapid in producing its effects, it is much more persistent. Nitro-glycerin is best given in the 1 per cent. alcoholic solution, beginning with minim doses; the tabellæ may also be employed. Nitrite of sodium is given in 1 to 2 grain doses, and erythrol tetranitrate in  $\frac{1}{4}$  grain doses.

In addition to dilatation of the vessels, fall of arterial pressure, and acceleration of the pulse, which all these drugs produce, there is invariably an increased frequency of respiration and a tendency to the formation of methæmoglobin. The presence of glycosuria has occasionally followed the administration of these remedies, and it is hardly necessary to add that the whole nervous system undergoes some reduction of irritability.

As regards the practical question of the comparative utility of the iodides and nitrites, it must be frankly admitted that the latter are in every respect less useful than the former.

In very many cases of cardiac pain, more particularly in elderly people, the use of opium is absolutely imperative. A dread of opium in cardiac disease has lurked in the minds, and has been expressed in the writings of many authors; in a large proportion of cases, nevertheless, more particularly of senile heart disease, opium appears to have almost a tonic effect upon the heart. It may be administered in a large variety of different ways, and combined with other remedies in accordance with the special needs of individual cases. Morphine is in most cases more valuable than opium itself. Opium and its derivatives, in addition to putting an end to cardiac pain, have a slight effect in increasing blood pressure, and in increasing the activity of the heart.

Chloral reduces the activity of the heart and the excit-

ability of the vasomotor centres. It is, however, of comparatively little importance in the treatment of serious cardiac pain, yet, in addition to other remedies, it is occasionally of service through its hypnotic and sedative effects.

Of the bromides, practically the same remarks may be made as of chloral, but there are many cases in which minor degrees of uneasiness may be relieved by their administration in combination with cardiac tonics.

Phenazone has been strongly recommended by Sée, and its employment in the minor forms of cardiac pain is often followed by excellent results.

Coca, in one of the many forms available, may be usefully exhibited as a means of producing a feeling of comfort, or, it would perhaps be better to say, of lessening the feeling of discomfort; while it, at the same time, certainly has some tonic effect on the circulation. The drug does not, however, possess in any degree the power of alleviating cardiac pain.

The treatment of toxic angina pectoris must have as its basis the prevention of further poisonous influence, and the removal of the effects which have been produced. The further use of the deleterious agent must be entirely prohibited, and every means taken to stimulate metabolism. Here will be found a not only legitimate, but most successful sphere for baths and exercises. Drugs are most uncertain in their effects. Even in cases presenting dilatation, digitalis and strophanthus are not of conspicuous utility, and the nerve excitants, more especially strychnine, are of much more value. The element of time is required, and much patience on the part of both the patient and the physician is demanded. This can easily be understood when it is considered that an intimate union of the poison and the protoplasm has taken place.

In order to meet the indications furnished by the reflex variety, all sources of irritation must be removed. The first duty is therefore to search for the cause of the disturbance. If this can be ascertained and eliminated, the anginous effects will tend to a natural termination, but recovery will be hastened by the use of the bromides. Vasomotor angina

pectoris appears to receive, as would naturally be expected, most benefit from the use of the nitrites. No practical experience of this form has been allowed me. Neurasthenic and hysterical types require to be treated on the general principles applicable to the morbid state by which they are conditioned.

It need hardly be added that in the case of such toxic conditions as gout specific remedies are required.

During an access of angina the prompt employment of appropriate means is required. Of remedies suitable for such an occasion, iodide of ethyl, given by inhalation, is certainly, in my opinion, one of the most important. Its action is probably due to the liberation of free iodine, which is rapidly absorbed by the blood. The chief disadvantage which has attended its use in my hands has been the occurrence of severe headache after it has been exhibited.

Nitrite of amyl, administered by inhalation, is the standard remedy of modern times for such paroxysms, and, given in a sufficient dose to produce its physiological effects, vascular dilatation, fall of arterial pressure, and cardiac acceleration, it is, without a doubt, in many instances wonderfully useful in cutting short the paroxysms. There are, however, as has been known from the earliest days of its employment, many cases in which it is absolutely useless. Isobutyl nitrite has, during the last two or three years, been employed with very considerable benefit. It is as yet impossible to compare its value with that of amyl nitrite; their action must be nearly identical, but their effects no doubt vary in degree.

Many attacks of cardiac pain are only amenable to the administration of chloroform or ether. In most instances the former is more serviceable from every point of view; but in some cases, where the condition of the circulation is extremely feeble, ether is certainly a safer drug.

The subcutaneous injection of morphine in a certain proportion of cases is absolutely necessary, and its effects may be beneficially increased by giving it in combination with atropine.

The cases which follow are selected so as to give examples, as far as possible, of angina pectoris of different types.



CASE 57. *Angina Pectoris with Aortic Valve Disease and Coronary Lesions.*—D. B., aged 54, ship-carpenter, was admitted to Ward 22 of the Royal Infirmary on 21st March 1894, complaining of pain coming on in paroxysms at the pit of the stomach, and radiating into both arms. There were no special hereditary tendencies to disease. The patient's parents died in advanced life from causes about which he did not know much, excepting that neither had suffered from anything like his own symptoms. His own brothers and sisters had always been healthy, and he himself was the father of sixteen healthy children. His social conditions had always been excellent, but his work involved considerable physical stress. He was perfectly temperate as regards alcohol, but he chewed a quarter of a pound of tobacco per week. His previous health had always been good, and he had in particular suffered from neither rheumatic nor specific disease. Eighteen months before admission, he observed that, on walking after taking food, he was attacked by burning pain in the epigastric region, which came on suddenly, lasted for about fifteen minutes, and abruptly disappeared. Three months before admission the pain increased in severity, and became almost constant.

The patient presented a healthy appearance as regards colour, but his face denoted some anxiety. His tongue was clean, and the digestive functions were satisfactorily performed, with the sole exception of some flatulent distension. The patient complained of a pain deeply seated in the epigastrium, whence it passed upwards in the direction of the throat, and radiated downwards from the elbows to the fingers, more especially to the ring and little fingers of both hands. This pain, although almost always present to some degree, became much worse at times, and the paroxysmal attacks were followed by distressing palpitation. The radial arteries, and, in fact, most of the superficial arteries throughout the body, were rigid and tortuous; the pressure was only moderate, and the vessel by no means full. The pulsation was regular, and its rate varied between 104 and 116; the individual beats were of a bounding character. On inspection there was considerable heaving of the præcordia, and the apex beat was visible at a point further out and lower down than usual. There was a considerable amount of bounding movement in the arteries of the neck. On applying the hand to the præcordia the impulse was found to be forcible and diffuse, but the apex beat was determined to be in the sixth space 5 in. from mid-sternum. No thrill was present. The area of cardiac dulness was considerably enlarged, reaching the upper border of the third costal cartilage above, and extending  $2\frac{1}{4}$  in. and  $5\frac{1}{4}$  in. respectively to right and left of mid-sternum. On auscultation two loud murmurs were heard at the base. A systolic murmur was distinctly heard at the right border of the manubrium sterni, about half-way up, and was propagated into the carotid and subclavian arteries; a diastolic murmur was audible in mid-sternum about the level of the fourth costal cartilages, which was conducted most distinctly down to the extremity of the xiphisternum. The mitral and tricuspid areas only showed a slight blurring or muffling of the first sounds, and the pulmonary area gave rise to no abnormal phenomena whatsoever.

The diagnosis in this case was that of a degenerative change in the walls of the aorta, producing obstruction of the orifice, incompetence of the valve, and interference with the coronary arteries, followed by cardiac dilatation. The patient was kept absolutely at rest, and his diet was so arranged as to cause the least distension of the hollow viscera possible. He was at the same time treated medicinally by means of iodide of potassium, and the solution of arseniate of sodium, while iodide of ethyl and amyl nitrite were recommended for the paroxysms of pain. For two or three weeks the patient appeared to improve, but on the evening of 24th April an excruciating attack of pain occurred. When seen, the patient was found to be sitting upright in bed with wild staring eyes, and an expression of great anxiety upon his face, which was perfectly blanched, and bedewed with a cold perspiration. The pulse was at this time 104, and possessed moderate volume and pressure. The patient received a hypodermic injection of one-third of a grain of morphine hydrochlorate, and as both amyl nitrite and ethyl iodide failed to produce any relief, he received some chloroform by inhalation. In spite, however, of all these remedies the agony continued, and the patient expired.

The post-mortem examination was performed on the following day by Dr. Leith, and the report states that both rigidity and lividity were present. On removing the heart, the pulmonary valve was found to be competent, but the aortic was markedly incompetent. The cone diameters were:—Pulmonary, 1.23; Aortic, 1.0; Tricuspid, 1.85; Mitral, 1.5. The pulmonary valves were quite healthy. All three aortic cusps were thickened. Their free margins were smooth, but thick. There were, however, two spots of early vegetations on one of the cusps. The orifices of both coronary arteries were atheromatous and largely blocked; both showed pale clots in their orifices. There was a ring-like band of atheroma well marked around the whole aorta at that level. The coronary arteries were but little, if at all, affected. The disease had invaded their orifices only so far as it was a lesion of the aorta, and not of the coronary arteries. The left ventricle was considerably dilated, and its wall relatively thin. It was about  $\frac{3}{4}$  in. thick at its base, and less than  $\frac{1}{4}$  in. towards the apex. The length of the cavity was  $3\frac{7}{8}$  in., the wall of the left ventricle was distinctly fatty. The right ventricular cavity was also dilated, its wall was more markedly fatty than that of the left. Both mitral and tricuspid valves were healthy. The aorta was atheromatous in diffuse scattered patches, chiefly showing a fatty change. Both lungs were œdematous; the right weighed 1 lb. 15 oz., the left 1 lb. 13 oz. The peritoneum was healthy; the liver weighed 4 lb. 4 oz. The gall bladder showed œdematous thickening of its walls, and contained some brown bile. The liver substance showed slight venous congestion. The spleen, which weighed  $7\frac{1}{2}$  oz., was healthy. The kidneys were deeply congested, the Malpighian bodies being especially prominent. The right kidney weighed  $6\frac{1}{2}$  oz. and the left 6 oz. The stomach was healthy, and the intestine, although practically healthy, showed some atrophy of Peyer's patches.

This may be regarded as a very excellent type of the fatal angina pectoris often associated with aortic affections.

CASE 58. *Angina Pectoris and Aortic Disease*.—K. K., aged 37, hawker, was admitted to Ward 22 of the Royal Infirmary, 26th February 1894, complaining of pain between the shoulders, palpitation, and breathlessness. His father had died at the age of 62 of some cause unknown; his mother died at the age of 48 of some form of rheumatism. About the other members of his family, most of whom had lived in Germany, his native country, he was able to give little information. His social conditions had been fairly satisfactory. He had suffered from rheumatic fever thirteen years previous to admission, but he had, as he believed, entirely recovered from it. In 1891 he began to suffer from palpitation and giddiness, but not to such an extent as to necessitate giving up work. He was in Ward 26 during 1893 for a fortnight on account of cardiac troubles. The attack for which he came under treatment began ten weeks before admission, in consequence of getting wet and being chilled while pursuing his usual avocation.

On examination the patient presented a somewhat pallid appearance, with a small spot of dusky red upon each malar prominence. His expression was always somewhat anxious, and at times peculiarly apprehensive. He complained of some dyspeptic symptoms, but the tongue was clean; there was no dilatation of the stomach, and the liver only reached the costal margin. The blood and glands presented no features worthy of record.

The pain, which formed the predominant symptom of which the patient complained, was always present as a vague uneasiness felt as if behind the sternum. If exposed to cold or damp, or if engaged in any physical or mental exertion, it sometimes became an overwhelming sensation of agony, situated, so far as he could define it, behind the sternum, but radiating outwards laterally in both directions, so as to present a somewhat irregular semicircle upon the front of the chest. It was also experienced at the back over an oval-shaped area situated mostly to the left of the vertebral column, but with almost a fourth to its right. These two areas were continued on to the arm, and the pain surrounded

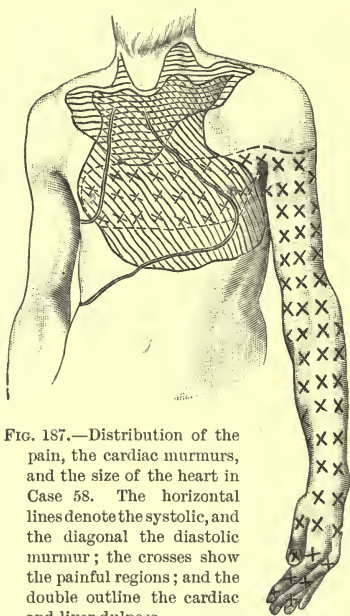


FIG. 187.—Distribution of the pain, the cardiac murmurs, and the size of the heart in Case 58. The horizontal lines denote the systolic, and the diagonal the diastolic murmur; the crosses show the painful regions; and the double outline the cardiac and liver dulness.



and passed down the whole of the arm and forearm from a little above the attachment of the deltoid to the tips of the fingers. The areas are shown in Figs. 187 and 188. The pain was of a sharp, lancinating character, and was accompanied by faintness, breathlessness, and palpitation. All of these were worse on exertion and excitement, and during one of the attacks, about a year before admission, he had fainted entirely away. On testing the areas over which pain was experienced, they were found to manifest considerable hyperæsthesia, and in front of the chest especially, there was exquisite tenderness. The pulse was 72. There was no alteration in the arterial walls, but the vessel was poorly filled; the pulsation was regular and equal,

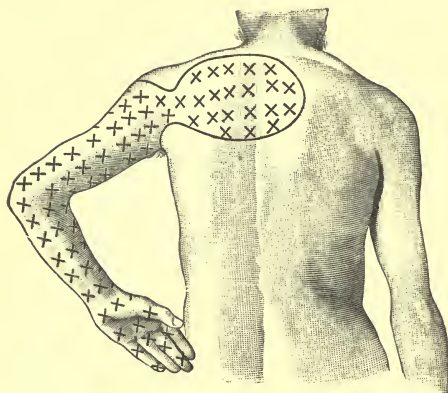


FIG. 188.—Area of pain and tenderness from behind.

but the pulse was short, sharp, sudden and collapsing. It could not be ascertained that there was any alteration in the pulse pressure on the occurrence of the pain. There was violent pulsation of the carotid arteries in the neck, and the apex beat, situated in the sixth intercostal space  $4\frac{1}{2}$  in. from mid-sternum, was forcible, but short in duration. On percussion the heart extended 2 in. to right, and 5 in. to left of mid-sternum. On auscultation two murmurs were heard at the base, with their greatest intensities near the aortic cartilage, and a somewhat wide area of propagation, which can be seen in the illustration, Fig. 187. In the mitral area there was simple impurity of the first sound. There was accentuation of the pulmonary second sound, and the tricuspid first sound was somewhat muffled. There were no features worthy of note in connection with any of the other systems.

There could be no doubt that in this case there was a genuine angina pectoris connected with disease of the aortic orifice. It is of interest to notice that this was probably of rheumatic, not degenerative origin, but the fact of his having suffered from acute rheumatism some years before, does not necessarily negative the possibility of some degeneration of the aortic and coronary walls, of which, nevertheless, there was no proof in the condition of the radial artery.

The patient was treated with moderate doses of iodide of potassium, along with some digitalis. He was for some days confined absolutely to



bed, but afterwards was allowed to get up, and move about the ward. In less than a month all the subjective sensations had disappeared, and even walking sharply outside of the hospital produced no feeling of pain. At his own special request, he was therefore allowed to return home. He reported himself to me from time to time at the Infirmary, and, continuing to take the iodide, all went well. In a few weeks, however, he sought readmission on account of a return of the pain from which he had previously suffered, and once more entered the ward. It was found that there was almost no pain in the præcordia or in the arm, but that he suffered from excessive pain and exquisite tenderness over the left half of the nape of the neck corresponding to the distribution of the third cervical nerve according to Head. The iodide treatment was persevered with, but did not seem to have much power of alleviating the symptom. Nitro-glycerin and nitrite of amyl were also employed without any noticeable benefit, but the symptom yielded rapidly to phenazone. It remained a knotty point, the solution of which could not be determined, whether this pain was of cardiac origin, or whether it was not a form of neuralgia. As soon as the pain had quite disappeared, he begged to be allowed to go home, and, on receiving permission to do so, he left the hospital.

Having heard nothing of him for some weeks, it occurred to me one day, when in the vicinity of his abode, to inquire how he had got on, when the information was vouchsafed to me that, on the evening of the day on which he left the Infirmary, he had gone to bed feeling particularly well, but, within half an hour of lying down, had suddenly leaped from bed with the cry that he was dying, and expired instantly.

CASE 59. *Aortic Disease with Angina Pectoris*.—G. M., aged 54, engaged in hawking, formerly employed as a ship-carpenter, came under my care in the Royal Infirmary, 10th September 1894, on account of pain in the chest. His father was drowned at sea when 60 years old. His mother died aged 50, and the patient stated that the cause of death was "the change of life." He had eight brothers and sisters, who were all well. He was the father of eight children, who were all, as was also his wife, in good health. His social conditions had been on the whole satisfactory, but when employed in shipbuilding he had undergone severe toil for twenty-five years. He had never been guilty of alcoholic excess, and had never had any venereal affection. The patient stated that he had suffered from smallpox, malaria, and scurvy. For three years he was engaged on one of the Indian troopships, and underwent great changes of temperature, varying between that of the refrigerator and that of the tropical sun. He had suffered from lumbago, and from pain in one of the great toe-joints. Nineteen months before admission he was attacked by severe pain in the chest, which darted up to the right shoulder and down the right arm. Treatment brought about considerable relief for a time, but the pain again returned, and attacked the left shoulder and arm, as well as those of the right side.

The patient, on examination, was found to be a man of 5 ft. 2 in. in height, and 7 st. 9 lb. in weight. Although slenderly built, he was

well developed as regards the muscles.

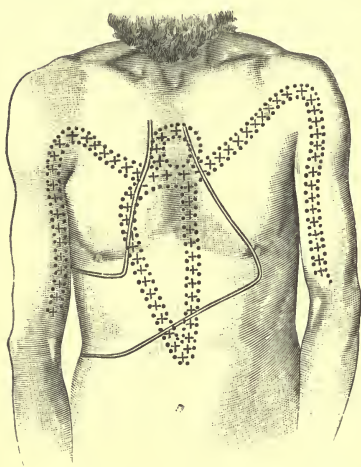


FIG. 189.—Cardiac and liver dulness and distribution of pain in Case 59, seen from before.

The face was deeply marked by smallpox. The teeth and gums were bad, many of the former being absent, and round the few relics of them which remained there was great sponginess of the gums. The tongue was flabby. The patient complained of flatulence and pyrosis, and stated that sometimes after taking food the pain immediately began. The hollow viscera were of normal size, and the liver extended from the fourth cartilage to the costal margin, occupying  $5\frac{1}{2}$  inches. The blood-forming apparatus showed no morbid phenomena. Any exertion, whether physical or mental, induced a paroxysm of pain, with a somewhat singular distribution. In the upper part of the præcordia the pain seemed to form a circle surrounding an area in which no uneasiness was

experienced, and from this circle it passed both downwards and upwards—downwards to form the margins, as it were, of a harp-shaped figure, and upwards as far as both shoulders, whence it passed down the inner side of the arms so as to reach the finger-tips. It also occupied each scapular angle. The areas are shown in Figs. 189 and 190. The pain was accompanied by severe palpitation and great dyspnœa, as well as giddiness. Such were his complaints of the worst kind of seizure. At other times the attacks were much less severe, the pain failing to reach further than the shoulders, and having no palpitation, dyspnœa, or giddiness. He was frequently seen in one of those paroxysms, and at such times the pulse was found to have a rise of pressure, the expression became anxious, the face turned pale, and beads of perspiration stood upon the forehead. Examination of the circulatory

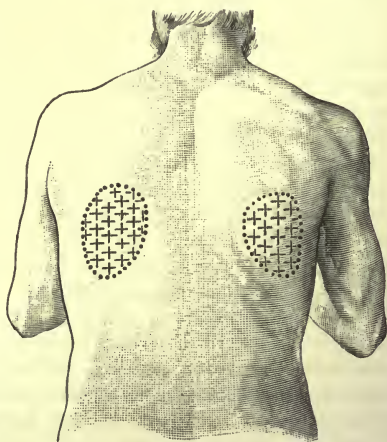


FIG. 190.—Areas of pain in Case 59, seen from behind.

organs showed that the radial artery was rigid and tortuous ; the vessel was full and its pressure somewhat high. The pulsation was regular, and usually about 70. The pulsations were somewhat quick, but not collapsing. There was a diffuse impulse in the fifth and sixth left intercostal spaces, and the apex beat was determined to be in the latter,  $3\frac{1}{2}$  in. from mid-sternum. The right and left borders of the heart were  $2\frac{1}{2}$  and 4 in. from mid-sternum ; the upper border was at the superior margin of the third left costal cartilage. On auscultation in the aortic area there were systolic and diastolic murmurs. In the mitral and tricuspid areas systolic murmurs were audible, and the pulmonary second sound was considerably accentuated. The respiratory system only showed some scattered crepitations over the base of the left lung, and there were no morbid appearances connected with any other system of the body.

The patient was obviously suffering from cardiac failure consecutive to arterial degeneration and aortic lesions. He was treated by means of nitro-glycerin in combination with digitalis, and amyl nitrite for the paroxysms. By the use of these remedies he considerably improved, and was dismissed at his own request.

This must be regarded as another instance of angina pectoris from aortic changes, probably affecting the coronary arteries, followed by myocardial effects.

CASE 60. *Angina Pectoris probably from Coronary Changes.*—J. W., engineer, aged 46, had consulted me on account of pain in the chest from time to time at the Royal Infirmary, and was kindly brought to see me there by Dr. Jamieson, 5th June 1898. His father had died at the age of 72, and his mother at 71 from causes unknown to the patient. Four brothers were alive, and well ; one died accidentally when an inmate of an asylum. Three sisters were in excellent health, and one was a patient in an asylum. The patient had enjoyed good health all his life, with the exception of a few unimportant illnesses, but he had passed through the stress of much hard work, and exposure to different climates. For some years he had suffered from pain in the chest and left arm. This, on careful investigation, was

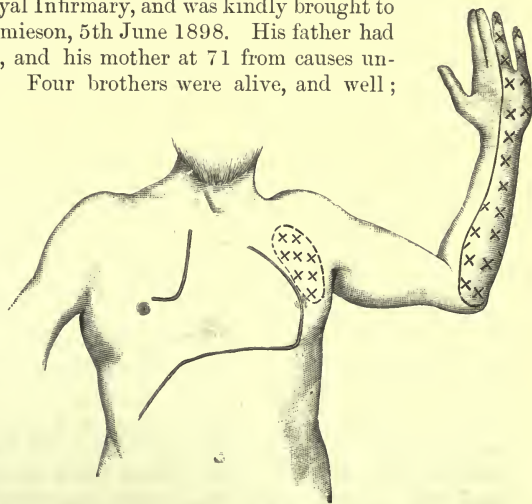


FIG. 191.—Distribution of pain in a case of angina pectoris probably due to coronary sclerosis (Case 60). The areas marked by crosses indicate the painful and tender regions.



found to be confined to an oval-shaped area in the infra-clavicular and mammary areas of the left side, and to extend from above the elbow on the inner side of the left arm, down to the tips of the ring and little fingers. The distribution is shown in Fig. 191. It corresponded, in short, to the distribution of the anterior thoracic and ulnar nerves. While the pain was present, hyperæsthesia of the painful areas was usually experienced, but, on the date mentioned, when the pain was almost entirely absent, it was found that these areas were less sensitive than the corresponding spots on the right side.

The arterial walls were somewhat thickened and slightly rigid. The radial artery was moderately full, and of fair pressure. The pulse rate was 88, its rhythm slightly irregular, and the pulsations rather unequal. No abnormal appearances were visible in connection with the neck or chest, and the apex beat could not be seen on account of the well-clothed condition of the patient's chest. It was felt, however, in the fifth intercostal space, somewhat diffuse in character, and with its chief intensity 4 in. from mid-sternum. The right border of the heart was 2 in., and the left  $4\frac{3}{4}$  in., from mid-sternum. The upper border of the heart was at the superior margin of the third costal cartilage. In the aortic area, the second sound was accentuated, and at the apex the first sound was slightly blurred. No other abnormal characters could be made out.

This case presented every feature of angina pectoris in one of its milder forms, and the affection probably depended upon a slight degree of alteration in the coronary arteries. The use of iodide of potassium and the careful regulation of every habit brought about so much improvement that uneasiness was seldom experienced.

CASE 61. *Angina Pectoris from Arterio-sclerosis and Cardiac Enlargement.*—G. B., aged 60, joiner, entered Ward 22, 5th April 1894, on account of pain in the chest. His father had died at the age of 70 of some disease about which he could give no information; his mother, when he was very young, from causes also unknown to him. His only brother died of apoplexy; his only sister had always been perfectly well. His social condition had always been perfectly satisfactory, and he had led a temperate life. His previous health had been so excellent that he had, as he expressed it, "never been ill a single day." Three months before admission, when walking home one afternoon after finishing his day's work, he was seized by a violent pain in the chest, and every day subsequently until the date of his admission he had a recurrence of the pain. The patient was a remarkably healthy-looking man, with a clear complexion, ruddy lips, and bright eyes, but his expression was somewhat anxious, and at times even apprehensive. The alimentary and the hæmopoietic systems showed no obvious departures from health. The patient described the pain as beginning at the xiphoid cartilage, with a sensation as if his inside were being tightly



squeezed. From this point the pain spread upwards until close to the junction of the manubrium and body of the sternum, where it split into two parts, each of which darted outwards to near the shoulder, and proceeded down as far as the elbow. The distribution of the pain is shown in the illustration, Fig. 192. The pain was always accompanied by breathlessness, so that he had to pant for breath, and yet was almost afraid to do so. It was also attended by bursts of perspiration. The radial arteries were somewhat hard, and slightly tortuous. The vessels were full and the pressure was high. The pulse rate was 102; it was perfectly regular and equal. The pulse waves were somewhat tardy and sustained. There was perfect regularity and equality. The apex beat was somewhat diffuse, but it presented a point of maximum intensity in

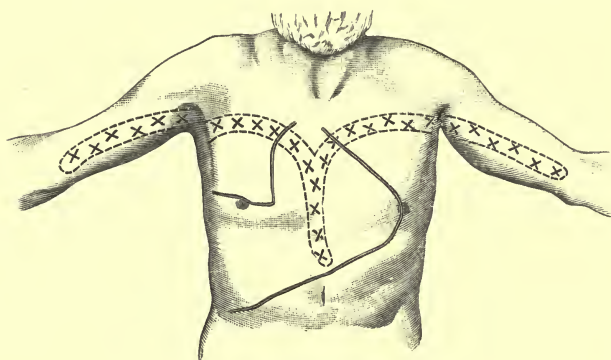


FIG. 192.—Distribution of pain in Case 61.

the fifth intercostal space, 4 in. from mid-sternum. It was slightly heaving in character, and was unaccompanied by any thrill, or any other abnormal appearance. On percussion the cardiac dulness reached  $2\frac{1}{2}$  in. and  $4\frac{1}{2}$  in. to right and left of mid-sternum. The aortic second sound was considerably accentuated. The mitral and tricuspid first sounds were low in tone and somewhat muffled in quality. The pulmonary second sound was of approximal normal intensity. No other abnormal phenomena were present. The patient was treated by means of iodide of potassium in infusion of digitalis, and absolute rest. In the course of a few days he lost every trace of uneasiness, and he was able to return home much relieved.

This case gives a good example of angina pectoris due to chronic degenerative changes in the heart and arteries, probably with coronary obstruction.

CASE 62. *Angina Pectoris*.—D. C., aged 70, a civilian retired from India, was seen by me along with Dr. Arthur Wilson on 19th January 1898 on account of severe pain in the chest. The patient's family history

was absolutely negative in regard to cardiac tendencies. His previous health had been most satisfactory in all respects, excepting for a few attacks of subacute fever in India. For about six months he had been complaining of a feeling of oppression in the chest, along with some uneasiness, amounting to actual pain, darting from the elbows to the tips of the fingers, no finger being more affected than the rest. He disregarded these feelings, however, and went about as usual. They gradually wore off, so that by the 15th January he was feeling as well as he had ever felt in his life. He had a good deal of exercise that day, and on retiring at night intended to have a hot bath, but, by some misadventure, the water was scarcely warm, and the bath which he took was almost cold. On the following day, which was Sunday, he got up early, and started after breakfast to walk to church, in connection with which he superintended the Sunday School. The church was situated at a considerable distance from his house, and great part of the road was uphill. During his ascent he was overtaken by a feeling of faintness, and feared he would never be able to reach church. This, however, he did, and performed his usual duties. Feeling tired, however, he remained quietly at home the rest of the day, and retired early to rest. About four o'clock in the morning he waked up from a sound sleep to find himself the victim of excruciating agony in the chest, situated more particularly at the lower portion of the sternum, whence it radiated upwards to both shoulders, and down the arms to the tips of the fingers. He at once sent for Dr. Wilson, who attended immediately, and administered nitrite of amyl by inhalation, along with alcohol and strophanthus. He obtained considerable relief by means of these remedies. Dr. Wilson enjoined absolute rest and easily digested food, while he persevered in the course of treatment which he had adopted.

When seen by me he was not suffering from any pain or uneasiness. His expression was placid, even cheerful, and the face betokened no circulatory disorder. The radial arteries were somewhat rigid and tortuous, as were also the temporals. The pulse pressure was not more than moderate, and the vessels were moderately full. The pulsation was perfectly regular, 70 per minute, but rather tardy. On inspection of the chest a slight flickering pulsation, auricular in rhythm, was observed at the lower end of each external jugular vein. The apex beat was invisible, but on applying the hand it was determined to be  $4\frac{1}{4}$  in. from mid-sternum in the fifth intercostal space. It presented no abnormality save a certain amount of weakness. No other impulse could be detected. On percussion the borders of the heart were 2 in. and  $4\frac{1}{2}$  in. to right and left of mid-sternum. On auscultation the cardiac sounds were feeble over both ventricles, but this was particularly so at the apex. The second sounds at the base were normal. There were no abnormal clinical features connected with any other system.

This patient showed evident arterial changes with some cardiac dilatation. The treatment adopted by Dr. Wilson was continued, but was from time to time modified according to

circumstances. Much improvement followed, and the condition has remained satisfactory to the present date.

CASE 63. *Angina Pectoris from Fatty Accumulation*.—A. M., aged 75, a widowed lady, has consulted me during the last few years, chiefly on account of breathlessness on exertion. Her family history was remarkably good, her father having lived to the age of 84, and her mother to the age of 83. All her brothers and sisters, with the exception of one brother and one sister, who died from affections unconnected with the circulation, were in the enjoyment of excellent health. The whole family had, however, suffered considerably from irregular gout in many of its protean forms. The patient was the mother of three daughters, two of whom were in good health, while the third suffered from some spinal symptoms due to an accident in early youth. The patient's previous health, until after middle life was passed, had been satisfactory, with the exception of one or two acute diseases from which she had suffered. About the age of 55 or thereby, she observed that she was apt to be breathless on exertion, and required to be somewhat more careful in regard to her health than had hitherto been the case. During the two or three years previously she had been more troubled in this way than formerly, and another symptom made itself manifest—a feeling of oppression in the chest, and pain running down the left arm.

The patient was of medium height, and decidedly plump. There was no arcus senilis, and the hair was very slightly gray. The tongue was clean, and the digestive functions were carried out most satisfactorily in all respects. The temperature presented a somewhat interesting course. It was frequently taken morning and evening for a length of time, and the average reading in the morning was 98°, while the evening was only 97.4°. The liver was somewhat enlarged, extending from the fourth cartilage to 2 in. below the costal margin, or 7 in. in extent. The spleen was normal in size. The patient was always somewhat breathless on exertion, and any slight exercise, as well as any mental excitement, was apt to produce uneasiness in the chest. This usually assumed the form of a feeling of tightness and weight in the præcordia, passing backwards to the shoulder-blades, and outwards to the left shoulder, but not infrequently of a painful sensation darting down to the left elbow, and sometimes reaching the little and ring fingers of the left hand. These sensations were usually attended by feelings of fluttering within the chest, attended by breathlessness. The radial artery was remarkably healthy, having but little tendency to rigidity, and absolutely no tortuosity. The pulse pressure was moderate, the rate 68. It was absolutely regular, and each pulse wave was somewhat tardy and sustained. One peculiarity of the pulsation arrested my attention—the amplitude of the wave underwent periodic alterations, becoming smaller in extent for a time and again resuming its former size. On examination of the præcordia no impulse could be detected, but on applying the hand, the apex beat was felt diffusely in the fifth intercostal space between 3 and 4 in. from mid-sternum. On percussion the margins of the heart were

2 in. and  $4\frac{1}{2}$  in. respectively from mid-sternum, and the heart reached the upper border of the third rib above. On auscultation in the aortic area the sounds were clear and distinct, the second, however, predominating. In the mitral area, the first sound was low in tone, and somewhat short in duration, and here the second sound was also more distinct than the first. In the pulmonary area, the second sound was somewhat accentuated. In the tricuspid area, the first sound was distinctly louder than in the mitral region, yet it was still much less distinct than the second sound. The examination of the lungs revealed no alteration, save comparative dulness over the left base posteriorly, at which point some crepitations were heard. There was no alteration in the breath sound, or vocal resonance in any part of the chest. The renal secretion was somewhat scanty. The skin acted very freely. The only implication of the nervous system was seen in disturbance of sleep, which was apt to be troubled during the night, while during the daytime a soporose tendency was often present.

The opportunity was afforded me on one occasion of witnessing one of the attacks of which she complained. The patient was sitting up in bed; the complexion was marked by what can only be called a livid pallor; drops of perspiration were hanging on the forehead, and anxiety was expressed in every lineament. The pulse was extremely irregular, and the heart very tumultuous.

No doubt has existed in my mind that this was an instance of angina pectoris caused by fatty accumulation invading the myocardium. By means of the occasional employment of nitro-glycerin or erythrol tetranitrate, along with strophanthus, often alternated with strychnine and arsenic, or colchicum and bicarbonate of potash, the disagreeable symptoms could be kept at bay.

CASE 64. *Slight Anginous Attacks from Tobacco.*—A. M., aged 24, student of medicine, consulted me, 13th December 1897, on account of pain in the chest, attended by considerable palpitation. His family history showed some tendency towards circulatory disturbances, as his father was a sufferer from a weak heart; his mother had died of some spinal affection. All his brothers and sisters were in excellent health. His social conditions had been excellent, and his previous health quite satisfactory. The symptoms for which he sought advice had commenced two or three weeks previously, whilst he was training for an athletic competition.

The patient was a tall, powerful, well-built man, with a remarkably healthy appearance. His height was 5 ft. 11 in., his weight 12 st. 2 lb., and his chest measurement 40 in. on inspiration. The tongue was slightly furred and rather tremulous, and the fauces somewhat congested, but there were no other symptoms of alimentary disturbance. The pain which was complained of was situated in the lower sternal region. It



was persistent, and was more a sensation of dull soreness than of sharp pain. It showed no tendency to radiate in the direction of either shoulder or arm, but it was attended by a sense of tightness and constriction across the whole chest. These sensations were invariably increased on exertion, and they were associated with considerable palpitation, which, although more frequent on exertion or excitement, had a tendency to appear spontaneously at irregular times. The arteries were perfectly healthy but rather empty, and the blood pressure was low. The pulsation was slightly irregular and unequal, its rate being 82. On examination of the præcordia, there was a diffuse pulsation over a wide area, yet the apex beat was quite distinct in the fifth intercostal space,  $3\frac{1}{2}$  in. from mid-sternum. The character of the pulsation was short and sharp. The area of cardiac dulness reached the upper border of the third left costal cartilage, and at the fourth it extended  $2\frac{1}{2}$  in. and 4 in. to right and left of mid-sternum. On auscultation the first sound was everywhere weak by comparison with the second. This was more particularly observable at the apex, where the first sound was rather high-pitched in tone, ringing in character, and short in duration. There was no systolic murmur in the tricuspid area even when the patient lay on his back, and the first sound was rather more distinct than at the apex. The second sound in the pulmonary area was considerably louder than in the aortic. There were absolutely no abnormal symptoms connected with any other system.

On investigating the habits of the patient, it was found that he was addicted to smoking heavily, and there was no doubt in my own mind that the features were those of a commencing tobacco angina. He was recommended to desist entirely from training for some time, and to renounce tobacco, while small doses of strophanthus and nux vomica were prescribed. In less than a month all the symptoms had disappeared, and the patient was restored to his usual health.

The case may be regarded as a most typical example of the cardiac pain which is characteristic of the excessive employment of tobacco, in one of its mildest forms.

CASE 65. *Severe Anginous Attacks from Tobacco.*—D. M., a medical man, aged 28, consulted me several times during the year 1896 on account of pain in the chest, and breathlessness on exertion, accompanied by palpitation. His family history was absolutely satisfactory, while his previous health and social conditions left nothing to be desired. The present attack had begun a few weeks previously, with a sense of weight and constriction in the chest, which gradually assumed the proportions of pain, for which he sought advice.

His general appearance was healthy, save for a slight degree of bloodlessness. The alimentary system called for no remark. The pain, of which complaint was made, was situated in the lower sternal region, with a tendency to pass upwards and to the left, but it could not be said to reach higher than the second left intercostal space. It was by no means

sharp, and was rather a feeling of a dull weight, accompanied by tightness; but on exertion it became more severe, and was accompanied by violent palpitation and considerable breathlessness. One interesting point was that these symptoms not only followed exertion, but often arose after meals, and sometimes had caused him to awake during the night by occurring spontaneously. He was seen by me on many occasions, and it was easy to determine that considerable alterations in the physical signs were present. The arteries were absolutely healthy and moderately filled, while the blood pressure was low. The pulsation of the radial artery was exceedingly variable; while it would at one time be beating regularly and evenly at a rate of 74, at another it would be irregular and uneven, with a rate of more than 100. There were no abnormal appearances on inspection of the neck and præcordia. The apex beat occupied the fifth intercostal space, and was by no means diffuse. On palpation it was determined at a point  $3\frac{1}{4}$  in. from mid-sternum. It was very sharp and badly sustained, but unaccompanied by any thrill. The cardiac dullness extended to the upper edge of the third costal cartilage, and its borders were 2 in. and  $3\frac{3}{4}$  in. to right and left of mid-sternum. On auscultation the heart sounds, like the pulsations of the arteries, underwent considerable variations. At one time they might be heard regularly and evenly with no modification of the sounds, save that in the mitral area the first sound was high-pitched, sharp, clear and ringing, while in the tricuspid region it was slightly muffled, and the second sound in the pulmonary was considerably louder than in the aortic area. At another time the sounds were extremely irregular, a succession of them occurring with great rapidity, so that they appeared almost to run into each other, the series being followed by a longish pause, and several pulsations separated by longer periods from each other. These alterations in the sounds coincided with the modifications in the pulse above mentioned. There were no symptoms connected with the other regions of the body, and there were no objective abnormal phenomena on examination, save some tremulousness of the muscles.

The patient in this case had been in the habit of sitting up very late at night, and smoking a large amount of tobacco while at work. In this instance, both *strophanthus* and *digitalis* were found to aggravate the symptoms, but the patient rapidly improved under the use of strychnine three times a day, along with some bromide of ammonium at night, followed after the lapse of two or three weeks by the administration of arsenic.

The case was a more severe variety of tobacco angina than that which has just been described.

CASE 66. *Hysterical Angina Pectoris*.—A. C., aged 45, housewife, was admitted to Ward 27 of the Royal Infirmary, 22nd January 1898, on account of an attack of acute bronchitis. The patient's father was alive and in good health; her mother had died of consumption at an early age. A sister had also fallen a victim to phthisis, and a brother died

at the age of eleven days with rather ill-defined symptoms. One brother and one sister survived, and were in excellent health. Her social condition had always been good, and her general surroundings perfectly satisfactory. She had passed through most of the usual children's diseases, and a point of much interest lay in the fact that when five years old she had suffered from an attack of dropsy, for which she had been tapped. Of importance also were the facts that when sixteen she had passed through scarlet fever, and that when about twenty she had been confined to bed for three months with acute rheumatism. From that time she had never been entirely free from breathlessness and palpitation. She had a second attack of acute rheumatism four years afterwards, which laid her up for two months, and a third attack fifteen years later, by which she was confined about the same period. In the month of March 1897 she had been troubled with swelling of the lower extremities and of the body, attended by severe præcordial pain, and a high degree of breathlessness. During the ten years previous to her admission she had been subject to pain in the præcordia, which varied greatly in severity and frequency, no attack occurring for months at one period, while at another time attacks were very frequent. It is unnecessary to recite the facts which led to the breakdown for which she sought admission. Suffice it to say that during a heavy washing she was exposed to cold, and bronchitis ensued. It was attended by great swelling of the legs and abdomen, and as it was difficult for her to obtain sufficient rest and attention at home, Dr. George Dickson recommended her to the ward.

The patient was found to be a strongly-built, well-nourished woman. Her complexion was somewhat dusky, the eyes looked suffused, there was dilatation of the veins of the cheeks and nose, and the lips were cyanotic. The fingers were distinctly clubbed. There was considerable œdema of the legs, arms, and abdomen. The attitude on admission showed considerable disturbance of the circulation, and the patient was unable to assume the recumbent posture. The temperature for some days after admission ranged between  $101^{\circ}$  and  $103^{\circ}$ . The pulse at the same time was usually about 100.

There was almost no appetite, but considerable thirst. Constipation had been the general rule during the past, but there were no other abnormal symptoms connected with the digestion. The abdomen was very prominent, but this was on account of plumpness; all the abdominal organs were apparently healthy. The liver extended from the fourth rib to the costal margin, and the lower border of the stomach was midway between the umbilicus and the xiphisternum. The whole glandular system was normal, and the spleen reached the mid-axillary line.

The patient complained of frequent attacks of stabbing pain in the region of the apex beat. This pain had always been worse after exertion or excitement, but had frequently awaked the patient during the night. The pain reached the left shoulder and shot down the left arm, producing a tingling sensation in the fingers. Each attack only lasted, as a rule, for a few minutes, and was not accompanied by any sensation of faintness, but a considerable degree of palpitation attended



the pain, and a profuse perspiration was almost always present during an attack. Breathlessness was more or less constantly present, and was invariably worse on exertion. There was hyperæsthesia over the region of the apex beat and in the infra-clavicular region, as well as down the arm, but of greater interest was the fact that firm pressure over the region of the apex beat produced a sensation of faintness. If greater pressure were employed, a sharp pain shot through the chest. The patient's eyeballs were turned upwards, and the upper lids drooped so as to nearly cover the globes, while an expression of almost fatuous content spread over her face. The patient expressed her belief that she could not open the eyes, and certainly remained in a somnolent condition for a considerable time, during which it was difficult to elicit any response to stimuli. There was dilatation of the superficial veins over the left side of the chest. No pulsation of any kind could be seen in the præcordia, but on palpation the apex beat was found in the fifth left intercostal space,  $4\frac{1}{2}$  in. from mid-sternum. The impulse was sharp and forcible, but not sustained, and it was markedly irregular in force and rhythm. It was accompanied by a well-marked presystolic thrill. The pulse rate was 76 to 80 after the bronchial attack passed off. The walls of the artery were slightly thickened, and the vessel was fairly full, while the pressure was moderate. The pulsation underwent remarkable cyclical changes, the vascular contents, the blood pressure, and the size of the blood wave undergoing a periodic increase and decrease. The upper border of the heart reached the upper edge of the third rib; the right border was  $1\frac{3}{4}$  in. and the left border 5 in. from mid-sternum. There was a rough systolic murmur in the aortic area, which almost entirely replaced the first sound, and was conducted upwards into the carotid arteries. The second sound was markedly accentuated. In the mitral area a high-pitched and rough presystolic was followed by a softer and lower pitched systolic murmur, along with the latter of which could be heard the first sound. In the pulmonary area a blowing systolic murmur could be heard, which was found to be propagated up from below. The second sound was weak. In the tricuspid area there was an apparent reduplication of the first sound, immediately followed by a systolic murmur.

It would be tedious to recount the various symptoms and physical signs connected with the lungs. It is only necessary to state that on admission there was a pretty sharp attack of subacute bronchitis. There were no symptoms connected with the integumentary system. The renal secretion was copious, varying from 42 to 72 oz. Its specific gravity was 1015, and its reaction acid. It contained no abnormal constituents. There was no symptom which might be considered as heralding the menopause. The nervous system appeared to be quite stable. The only change as regards the sensory functions consisted in a frequent headache over the front and top of the head, while the sight had been becoming somewhat dull during the four years previous to admission. There were neither anæsthetic nor hyperæsthetic regions, with the exception of that which has been mentioned at the apex of the heart. The motor and reflex functions were in no way modified, and the vasomotor and trophic were



likewise healthy. There was absolutely no evidence of any embarrassment of the cerebral functions.

On admission the patient was treated for the bronchitis by means of a tent into which steam medicated with benzoin was led. She also had stimulant expectorants, with some digitalis on account of the cyanotic tendency. By means of this treatment the bronchial attack entirely disappeared within ten days, while the dyspnœa and cyanosis had almost vanished. With the disappearance of the respiratory troubles, however, the subjective sensations in the præcordia became somewhat more violent. By means, nevertheless, of saline aperients, along with nitro-glycerine, considerable improvement was produced.

In this case there was, along with some degeneration of the arteries, an enlargement of the heart. The extent and form of the cardiac dulness seemed to me more characteristic of dilatation and hypertrophy—in the final stage of failure—from increased peripheral resistance, than of changes consecutive to mitral disease. Be that, however, as it may, there were distinct structural changes in the circulatory organs, with which might well be linked attacks of angina pectoris. There was no reason to doubt that the attacks were anginous, but, from the singular phenomena by which they were accompanied, the case seemed to me to fall very properly under the neurotic class.

## MOTOR AFFECTIONS.

The motor disturbances, which may be, and sometimes without doubt really are, of functional origin, have been fully considered from the semeiological point of view in an earlier chapter. The simple alteration in rate shown by bradycardia, or diminished, and tachycardia, or increased frequency, as well as the more complex appearances of tremor and delirium cordis, palpitation, and syncope, have been discussed as symptoms, while their association with certain of the morbid changes in various structural lesions of the heart, and the appropriate methods of treating them, have from time to time been mentioned in previous chapters. There remain for consideration two groups or syndromes of symptoms, which on account of their intrinsic importance require separate description.

### RECURRENT TACHYCARDIA.

The symptom-complex, now commonly recognised by this term, appears to have been first observed by Cotton, who was immediately followed by Edmunds, Watson, and Bowles, while further observations were made shortly afterwards by Nunnely, Cavafy, and Farquharson. The name tachycardia seems to have been introduced by Gerhardt, and the more distinctive title, paroxysmal tachycardia, was employed for the first time by Bouveret. Within recent years, besides many individual cases of the disease, some excellent studies of the affection have been published—among them, Pröbsting, Bristowe, Taylor, Larcena, Kirsch, Martius, Herringham, and Williams. The investigations of Martius and Herringham deserve special mention, on account of the care with which they were carried out, and the valuable conclusions which were reached.

ETIOLOGY.—Among the predisposing causes of this curious affection, hereditary transmission has been placed beyond doubt by an observation of Cettinger on its occurrence through four generations. With regard to sex, of 53 cases collected by Herringham, 30 occurred in men and 23 in women. In 40 of these cases the ages were definitely ascertained, and of those 7 had begun to suffer from the affection in childhood, 12 between the ages of twenty and thirty, 13 between thirty and forty, 10 between forty and fifty, and 3 beyond the age of fifty. Previous diseases appear to exert some influence. Rheumatism, influenza, diphtheria, and measles have been observed in this connection, while the association of the affection with malaria has been described by Faisans.

The exciting causes are usually such as give rise to physical disturbance. A blow over the heart has been recorded, but sudden exertion and mental excitement are very much more common, while gastric disorders, no doubt by mechanical interference, sometimes produce the symptom.

MORBID ANATOMY.—Only six cases of this interesting affection have hitherto been examined after death; in one there was fatty degeneration of the heart muscle, in two there was chronic interstitial myocarditis, and in three there was

cardiac dilatation. In several other cases different cardiac lesions have been determined during life, such as diseases of the orifices and valves, or dilatation and hypertrophy of the myocardium, but the six instances referred to are the only ones in which a post-mortem examination was obtained. It is of interest to notice that no trace of any nerve lesion has ever been detected.

**SYMPTOMS.**—The essential nature of the affection lies in its recurrent and paroxysmal character. Attacks come on, as a rule, without any approach to periodicity, and their duration varies from a few minutes to several days.

Subjective sensations, such as uneasiness, or even pain, in the chest, numbness, or tingling, in the arm, palpitation, fluttering, or some other motor disturbance, are occasionally experienced by patients, and there is apt to be some disturbance of the function of sleep. There is rarely any considerable degree of breathlessness. The aspect of the patient is generally characterised by pallor, but cyanosis may be present. Some exaggerated pulsation of the carotid arteries in the neck may be visible, but, instead of this, a very evident jugular pulsation may be found.

The pulse becomes enormously accelerated. It is, on account of fluctuations in force, often difficult to determine its rate by the sense of touch, so that it may be necessary to estimate the rate by auscultation of the heart sounds. The arteries are in general rather empty, and the pressure is distinctly low. The rate may be far above 200 per minute, and the character of pulsation may be both irregular and unequal. It is an interesting point that during an attack the pulse maintains the same rate during the night and the day. On examination of the præcordia the area of cardiac pulsation is found to be enlarged, while the force is almost invariably diminished. The area of cardiac dulness almost always surpasses the normal boundaries, and the sounds, particularly the first, lose some intensity. Needless to add, in certain cases there are cardiac murmurs of different kinds.

Associated with those cardiac symptoms, there may be some pulmonary hyperæmia or subcutaneous œdema, or albuminuria.

The intrinsic nature of the affection must be purely a matter of inference. Tuchzek suggested the theory, which has since been widely accepted, that the condition is due to paralysis of the vagus. Irritation of the sympathetic system has also been brought forward by Nothnagel as a probable cause. The affection is termed by Debove and Boulay a bulbo-spinal neurosis; Talamon, on the other hand, suggests that the affection is of epileptic nature. There has never, however, been any proof in favour of any of these hypotheses. It need hardly be added that all such views are purely speculative. As already mentioned, fatty degeneration and interstitial myocarditis were present in half of the cases examined after death, and it seems to me that West is probably correct in his opinion that the myocardium is the seat of the lesion. Herringham observes that, if an implication of the nerve-endings is admitted, the facts of the affection seem strongly in favour of West's view. This is undoubtedly the best explanation of the condition. As is very well known, brief attacks of delirium cordis are common in myocardial degenerations, and paroxysmal tachycardia must be regarded as a somewhat analogous condition, differing mostly from it in respect of greater duration.

My own opinion as to the essential nature of paroxysmal tachycardia is, that it must be regarded as analogous to the respiratory changes grouped under the term Cheyne-Stokes breathing. It is impossible to start from any other standpoint than that of the automatic activity of the heart muscle, seeing that the cardiac ganglia and their connections are only means by which the cardiac movements may be modified. It has already been shown that periodic respiration is probably the result of an interference with the normal rhythm of respiration through malnutrition of its centre. One of the illustrations employed to explain this view was the observation of Steiner that *Medusæ*, when kept in sea water insufficiently renewed, showed remarkable modifications in the movements of the calyx, which fall into periodic groups separated from each other by pauses. The essential cause of paroxysmal tachycardia seems to be a periodic variation in the functional activity of the heart muscle, but it would be



unphilosophic to deny that some diminution or perversion of the tonic influence of the nervous system over the heart may be partly responsible for the condition.

DIAGNOSIS.—But little remark is called for in respect of this aspect of the subject, seeing that the determination of the affection rests upon such well-marked symptoms.

PROGNOSIS.—The outlook cannot be regarded as satisfactory. There is an undeniable tendency in this condition to become worse until it ends in death. Herringham points out that after 30 years of age a patient suffering from this affection is never safe from death, and he has analysed the cases on record with a view to determining the ages at which death has occurred. Before the age of 30 there have been only 2 deaths; between 30 and 40, 5; between 40 and 50, 5; and over 50, 4.

TREATMENT.—The treatment must necessarily depend in great part upon the conditions which have preceded the affection, as well as those which underlie it. If there be any discoverable evidence of cardiac degeneration, treatment must be adopted to arrest and obviate the process. The general lines upon which this must be conducted have already been sufficiently discussed.

If dilatation, apparently due to weakness, is present, the use of baths and exercise will be found to be of benefit. Pressure upon the thorax, as recommended by Rosenfeld, seems to be helpful in the attack itself. Pressure on the vagus nerve as it passes down the neck, seems occasionally to be useful, but it often fails. Naturally the remedies which might be tried would be those which have an influence over the rate of the heart, more especially digitalis, and its congeners; none of them can be claimed as producing any effects of real benefit.

CASE 67. *Recurrent Tachycardia*.—A. W. C., aged 71, fisherman, attended as an out-patient in the waiting-room of the Royal Infirmary complaining of faintness and giddiness.

So far as could be ascertained, there were no hereditary tendencies towards any particular disease. His occupation, that of a fisherman, not only exposed him to very considerable physical stress, but to great exposure to the weather. His surroundings otherwise were perfectly satisfactory. He was a native of, and had lived all his life in, Burra,

one of the south-western of the Shetland Islands. His previous health was unmarked by any morbid incidents of special importance; in short, he appeared to have been wonderfully exempt from illness of any kind. About ten years before his visit to the Royal Infirmary he had been complaining of attacks of giddiness, often passing into a sensation which was described as a "dwam," and this faint feeling often forced him to desist from any occupation in which he might be engaged. It was not attended by any uneasiness in the chest, and there was no breathlessness at any time.

The patient was a tall, well-built, muscular man, with a pale complexion. His gait was marked by a slight stoop, yet his movements were somewhat more active than might have been expected from his years. The lips were healthy in colour, as were the mucous membranes of the mouth and eyes. The temporal arteries were somewhat tortuous. The pulse, after he had sat quietly for a few minutes, was found to be 250 per minute. The artery was rather rigid and slightly tortuous, but the vessel was somewhat empty, and the pressure of the blood low, so far as could be made out on account of the alteration in the arterial walls. There was a slight flickering in the external jugular, and the area of cardiac pulsation was enlarged. The cardiac impulse was found to be diffuse and feeble. Percussion showed that the borders of the heart were to right and left 3 and 4 in. respectively from the middle line. The heart sounds were feeble, more especially the first sound in the mitral and tricuspid areas, and the sounds there were in strong contrast to the second sounds at the base, which, nevertheless, were by no means loud. The aortic and pulmonary second sounds were so similar in intensity that it was impossible to determine which was the louder. There were no symptoms of respiratory disturbance beyond breathlessness on exertion, and the physical examination of the lungs revealed no abnormality. The other systems of the body called for no remark.

The patient stated that he had been seized by one of his attacks on his way to hospital, and that he was exhausted and giddy while being examined. By the time the examination was concluded, he described himself as feeling better, and when the pulse was examined at that time, its rate was found to have undergone a great change, being much less frequent; in fact, when it was timed, it was found to be only 78 or 79 per minute. The artery was also fuller, its pressure higher, and the pulse wave larger. It was still perfectly regular, as it had been when first examined.

It was quite clear that this was a case of paroxysmal tachycardia, but its severity was by no means so great as has been commonly described by the various writers who have devoted attention to the subject. The production of the symptoms seemed to belong essentially to a condition of chronic myocardial change of a fibroid character, associated with arterial sclerosis.

He was recommended to enter the Infirmary for a short time in order to be carefully watched, but, as he preferred to remain outside and report himself later, he was treated by means of digitalis and nux vomica. Some days later he returned, stating that he was better, and that no paroxysm had recurred during the interval. He went northwards the following day. Since that time information has from time to time been sent to me, from which, so far as could be gathered, he continued in fairly good health.

CASE 68. *Fatty Infiltration with Paroxysmal Tachycardia*.—A. M., a lady aged 64, was seen by me along with Dr. Craufurd Dunlop, 1st December 1897, on account of extreme breathlessness. Her father had died from heart disease at the age of 85, and her mother at 87 from chronic bronchitis. There were two brothers and two sisters in good health; two brothers had died, when two years old, of measles.

The patient's health had been satisfactory until the spring before she was seen by me, when she passed through a troublesome attack of cardiac failure, since when she had never been quite so well as formerly. The patient was extremely plump, and, from deep cyanosis, presented a purple tint of face, with lips of the colour of a ripe blackberry, and a tint like that of the bilberry in the nostrils and lobules of the ears. Her hair was snow-white, and there was a well-marked arcus senilis in each eye. The tongue was clean, and the alimentary system showed no alteration, not even any enlargement of the liver dulness. The spleen and thyroid gland were normal in size. There was no opportunity of examining the blood. The pulse was usually between 110 and 120 when seen by Dr. Dunlop, but it went up to 150 and 170 at times. On the occasion when we saw her together its rate was 172. The vessel wall was but little altered. The artery was moderately full, and the pressure fair. The pulsation, although extremely frequent, was perfectly regular and equal, and it was impossible to make out any periodic variations in rate, rhythm, or size. On examining the neck and chest no pulsation could be seen, and on placing the hand over the præcordia it was impossible to ascertain whether any pulsation was present or not. The cardiac dulness extended 2 in. and  $4\frac{3}{4}$  in. to right and left of mid-sternum respectively, and the area of dulness reached the level of the third rib. Auscultation showed that the sounds were clear and ringing, but high in pitch, and short in duration, while at the apex the first sound was doubled.

The patient suffered much from orthopnœa, yet, on examining the chest, there was absolutely no sign of hyperæmia; not the faintest impairment of the percussion sound, or the slightest accompaniment of the breath sounds being elicited. The integumentary system, apart from its adiposity, was in all respects natural, and the urinary and nervous systems were intact.

There could be no doubt that in this case a considerable degree of fatty infiltration was present, and it seemed prob-

able that it had to some extent interfered with the action of the heart, probably from resulting degeneration. Dr. Dunlop kindly informed me that on his next visit he found the rate of the heart between 80 and 90, and on a subsequent occasion, when we again saw the patient together, the pulse was one or two beats below 80. The treatment adopted was absolute rest, light food, and strophanthus along with sal-volatile.

### RECURRENT BRADYCARDIA.

A condition in most respects the opposite of that which has just been discussed was first observed by Adams. The same condition subsequently attracted the notice of Smith, and Stokes studied it with great care. It has been adverted to by several writers since these three great Dublin physicians gave it prominence; but the writer who has, within recent times, most carefully studied the condition is Huchard, who has proposed to attach the names of Adams and Stokes to the complex of symptoms.

ETIOLOGY.—The affection is certainly more common amongst elderly people than amongst those who are young and middle-aged. The original patient described by Adams was 68 years old, and those of Stokes were aged 50 and 68; advanced years must be regarded as an important factor in the production of the condition.

Certain acute general diseases appear to have considerable influence; this was remarked by Stokes; chief amongst these is certainly influenza. Such causes are responsible for most of the cases coming on in early or middle life.

In addition to such causes, the various factors which give rise to the structural alterations, about to be mentioned, are necessarily of importance in the production of this group of symptoms. Such agents have already been fully described in the section dealing with myocardial diseases.

MORBID ANATOMY.—The lesions which have been most commonly observed in recurrent bradycardia have belonged to the class of myocardial degenerations. In the original cases described by Adams, Smith, and Stokes adipose changes were



prominent. It is rather difficult, on account of the comparative rarity of the affection, to have any statistics of value as to the relative frequency of different lesions, but it seems probable that fibroid changes are rather more frequent than alterations of a fatty character. The condition is especially associated with arterial sclerosis, and it is therefore only what might be expected that interstitial myocarditis should be often found. Huchard is strongly of opinion that sclerotic changes in the arteries of the brain, and more especially of the medulla oblongata, are constantly in association with the group of symptoms.

SYMPTOMS.—The principal features of this condition, which indeed overshadow the others, are connected with the condition of the nervous and respiratory functions, and complaints of giddiness, faintness, or even transient unconsciousness attended by breathlessness, lead to investigations which reveal the real nature of the case.

There is nothing characteristic in the general appearance of the patient, unless the attitude should betoken respiratory distress. Pallor is, however, usually present, but sometimes a considerable degree of cyanosis may obscure it. On observing the patient more closely, there will often be seen some changes in the temporal arteries. All the arteries are, as a rule, rigid and tortuous, but they are, for the most part, less well filled than should be the case, and the blood pressure is below the normal. In some cases the fulness and pressure are above normal. These appearances are, no doubt, produced by failure of a heart which has been previously hypertrophied, and the pulse condition is that which has been described by Broadbent as that of "virtual tension." The rate of the pulse is in most cases permanently reduced, and during the recurrent attacks it falls to a still lower rate. In a case of Halberton's it fell to 5. It is quite usual to find the pulse perfectly regular, but irregularity and inequality may be present. Cyanosis, as has been mentioned, occasionally occurs, and there may be cedema of the dependent parts. Different varieties of cardiac pain have been described, but many cases are entirely free from any uneasy sensations in the chest.

On inspection of the præcordia the apex beat, when

observable, has usually been found to be further out than in health. It yields very varying results on palpation, sometimes giving the impulse characteristic of hypertrophy, at other times that which is found in dilatation. The area of cardiac dulness is generally increased. The first sound may be, as in hypertrophy, long and low, or, as in dilatation, short and sharp. It is usually surpassed in intensity by the second sound, especially in the pulmonary area. The sounds may be obscured by the murmurs characteristic of different valvular lesions.

Dyspnœa is a common feature of this interesting complex of symptoms, and occasionally it may take the form of severe cardiac asthma. At other times well-marked Cheyne-Stokes respiration is present, and this is more particularly the case when the paroxysmal attacks are present.

The urine is often scanty. Even when renal cirrhosis is present in association with arterial sclerosis, the cardiac failure commonly present renders the amount smaller. Albumin and tube casts may be present.

Cerebral symptoms almost surpass all the others. During those periods of rare pulsation such brain symptoms are mostly seen. They manifest considerable variability. Sometimes there are merely sensations of giddiness and faintness, but at other times there may be lapses of memory, or even total unconsciousness; on the other hand, convulsive seizures have been described.

The starting-point of the complex of symptoms is to my mind clearly the heart. When the sclerosis of the arteries of the brain diminishes its blood supply, and when the heart lessens its activity, the quantity reaching the brain is too small to sustain the cerebral functions, hence the faintness, giddiness, unconsciousness, and convulsions.

Why the heart should manifest such infrequency of contraction is at present impossible to understand. Recurrent tachycardia is found in conditions closely resembling, if not identical with, those underlying the condition now under consideration, and by no reasoning can a valid explanation be evolved. It is futile to peer further at present into the gloom surrounding the subject. Until we know why such a disease

as influenza can cause in two patients, apparently closely resembling one another, tachycardia on the one hand, and bradycardia on the other, we must be content to leave this question unsolved.

Huchard attempts to explain the whole group of symptoms by arterio-sclerosis of the vessels of the hind brain, and appeals to the well-known facts of permanently infrequent pulse in lesions of the upper cervical part of the vertebral column in proof of his views. A permanent reduction of rate is, no doubt, common enough in such cases, but they have no paroxysmal attacks.

DIAGNOSIS. — Infrequency of the cardiac pulsations is common enough as the result of many different conditions, such as degenerative changes in the myocardium, and the effects of specific poisons, like that of influenza. Such symptomatic bradycardia must be carefully distinguished from the syndrome now under consideration, the essential feature of which is that, with a permanent diminution in the frequency of the cardiac impulse, there are paroxysmal attacks of still greater infrequency. In this lies the diagnosis of the condition, and it is therefore easy to determine its presence. It need hardly be added that the rate must be determined by the examination of the heart as well as the pulse.

PROGNOSIS. — Paroxysmal bradycardia is usually associated with grave structural alterations of the heart, and it is, for the most part, of evil prognostic omen. Cases, however, have been recorded of the affection coming on in the prime of life, and, under appropriate treatment, passing away. With the exception of a few cases of this kind, the group of symptoms now under consideration usually persists until death, which is commonly somewhat sudden.

TREATMENT. — The great aims to be set forth in the management of this group of symptoms are, while strengthening the heart, to diminish the peripheral resistance. All general measures which can increase the nutritive activity of the whole system must be adopted, and the lines of treatment are such as have been described in the chapter upon myocardial degenerations.

The drugs which are of the most importance are iodide of

potassium, and nitro-glycerin, continued over a considerable length of time. These drugs may be given in combination with general tonic remedies, and if cardiac failure should be a prominent symptom, they may be most advantageously associated with strophanthus, strychnine, and nitro-glycerin. The two last mentioned are strongly recommended by Morison. Digitalis has in my own hands invariably failed of any beneficial effects.

CASE 69. *Recurrent Bradycardia*.—A. M., aged 82, was seen by Dr. Balfour and myself, along with Dr. Marshall of Hamilton, on account of severe breathlessness and œdema. The family history showed a considerable tendency to arthritic and arterial lesions. Both parents had, however, lived to advanced years. An only brother had been cut off early in life with some infectious fever. One sister had died in advanced years of chronic bronchitis and cardiac failure, and another, also at a late period, of arterial degeneration and asystole. Two other sisters enjoyed tolerably good health, but one had a weak heart and a liability to syncope, while the other had well-marked arterial sclerosis. The patient was pale, with a tendency to lividity of the dependent parts, and there was slight œdema about the ankles. The pulse rate was usually a little above or below 60, but when seen by us it was 34, and it was stated to have frequently fallen to between 20 and 30. The arteries were hard and tortuous, indifferently filled, and with low pressure. There was well-marked venous pulsation in the neck. The apex beat was rather beyond the mammillary line. Its impulse was forcible, but slapping in character, and there were soft blowing murmurs of systolic rhythm, in both the mitral and tricuspid areas. The aortic second sound was loud and ringing, but it was surpassed in intensity by that in the pulmonary area. There was great dyspnœa, with a tendency towards the Cheyne-Stokes rhythm. The respirations were extremely deep. During the periods of diminished frequency of the pulse, the condition of the mental powers underwent great alteration, periods of dulness, if not complete obscuration, being present.

There could be no doubt that this case furnished a striking example of the condition under discussion. It was obvious that along with arterial sclerosis there was cardiac dilatation probably from fibrosis, succeeded by failure.

Digitalis had been tried in order to obviate the threatened asystole, but was attended by such distressing aggravation of all the symptoms that it had to be discarded. By means of ammoniacal, ethereal, and alcoholic stimulants, along with strychnine, some temporary relief was obtained, but in a few days the patient passed away in one of the attacks.



## CHAPTER XVI.

### DISEASES OF THE AORTA.

THE aorta is subject to structural alterations, similar to those which are found throughout the arterial system in general; these changes, however, although essentially the same in kind, vary considerably in degree, since they are influenced by the special structure of the aorta on the one hand, and the local conditions of blood pressure on the other. In the aorta it is possible to make out the three arterial coats, but these depart in some respects from the strata of one of the smaller arteries. The intima differs from the same tunic in other arteries, not only by being much thicker, but also by containing some muscle cells, more connective tissue, and elastic fibres arranged in laminae. The middle coat, instead of being almost entirely composed of unstriped muscular fibres, is for the most part formed by connective tissue and yellow elastic fibres; the latter form thick layers alternating with the muscle cells, and are embedded in delicate connective tissue, with a branching network of fine elastic fibres. The adventitia is very much thinner than in the case of the smaller arteries. It follows from these distinctive structural features that the aorta, while less contractile, is endowed with more resistance to stress, and greater elastic recoil after distension.

On account of its proximity to the heart, the aorta is subject to much greater vicissitudes of pressure than any other blood vessel in the body. It must, therefore, necessarily be especially liable to such affections as have their origin in excessive pressure.

There is another consideration to which attention must be

called. All parts of the aorta are not equally subject to the stress produced by the circulation. The greater curvature of the vessel receives the direct impact of the blood as it leaves the heart. The aorta presents at its root the three bulgings known as the sinuses of Valsalva, and, farther up, a transverse section shows an oval instead of a circular outline. This is caused by a bulging of the wall on its greater curvature, usually known as the great aortic sinus. It has often been regarded as really aneurysmal, but, as Cunningham has more particularly emphasised, it is found in the embryo, and cannot therefore be produced in this way.

It has been the habit of certain observers, as, for example, Quincke in his exhaustive monograph on vascular affections, to divide arterial diseases into groups, according as they have their origin in the external, middle, or internal tunic. Such analysis does not fall within the scope of the present work, nevertheless, some brief reference to this aspect of the subject may well be allowed a brief reference in these introductory remarks. Exarteritis, commencing with the adventitia, is occasionally met with in the aorta as a secondary process, consecutive to some acute process in the neighbouring tissues. As examples of this special affection may be mentioned the implication of the external coat of the aorta in pericarditis, mediastinal abscess, and ulceration of the trachea or œsophagus. Cases of this kind have been narrated by Sprengler and Leudet. The resulting lesions in the artery are sometimes characterised by an increase in the connective tissue with cellular infiltration, but the process may be so acute as to end in suppuration. The more chronic variety gives rise to thickening and adhesions to the adjacent structures; the more intense may lead to acute aneurysmal dilatation or to rupture.

Alterations in the structure of the tunica media are always secondary. An increase in the thickness of this coat may result from causes operative through continuous irritation from without, as in the hyperplasia which has just been mentioned, or from increased stress, as is sometimes found in aortic incompetence with cardiac hypertrophy; this latter point has been more especially described by Quincke. Atrophy and

degeneration of the media are, however, much more commonly observed. Their causes are as yet by no means thoroughly understood, but they seem undoubtedly in many cases to have their origin in hereditary tendencies, while long-continued overstrain and changes in the intima are the commonest existing factors. It is possible that some alteration in the vasa vasorum, such as will be referred to in the section on aneurysm, is intimately connected with the atrophic and degenerative processes.

The inner coat is that which is most liable to morbid processes; as it, however, is the main subject of the following pages, it is unnecessary to dwell upon it at present.

The evolution of our present views in regard to this subject is full of interest. While observations on certain of the lesions were made by Scarpa, the first serious attempt at a classification was that of Hodgson, who recognised three changes—cartilaginous, pulpy, and purulent. Craigie also described three types—calcareous, atheromatous, and steatomatous. Both these observers seemed to consider the lesions as deposits. Rokitsansky first recognised that the changes occurred in the intima, and resulted in two chief lesions, atheromatous and ossific, *i.e.* calcareous. Virchow finally placed the correct construction upon the nature of the process, by showing that it was due to an increase of the connective tissue normally present, and he accordingly introduced the term *endarteritis*.

Most of the lesions of the aortic walls are found in connection with arterial sclerosis, and, therefore, a few preliminary observations must be made upon this subject. Arterial sclerosis, undoubtedly, takes its origin in a condition of weakness and loss of elasticity. This is essentially a senile change, but it makes its appearance at very variable ages, according to the individual peculiarities of those subject to it. In some families it may occur in the third decade, or even in the second, whilst in others it never appears at all. As will be remembered, Harvey recorded that the celebrated Thomas Parr, who died at the age of 152, had arteries absolutely free from any structural change. To compensate for the loss of resistance and of elasticity, several different processes ensue.

In the first place, the intima undergoes considerable thickening, and this is commonly found to occur in the situations most exposed to stress. The media usually becomes also greatly thickened, while an increase in the adventitia is often to be seen. These changes result in an increase of resistance, or, as it may be termed, a tendency towards rigidity.

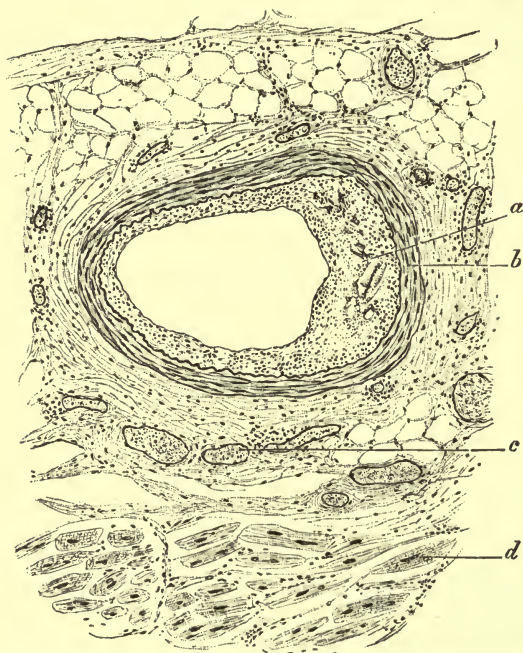


FIG 193.—Atheroma of coronary artery,  $\times 50$ . *a*, Greatly thickened patch of intima, with degenerative changes in the deeper layers; *b*, muscular coat; *c*, adventitia with engorged vessels; *d*, muscle fibres of the heart.

Thoma prefers to regard the result of these changes as producing an increase of elasticity, but in so doing he shows a misconception of physical terminology. The property of returning to the previous size and form after distension, to which alone the term elasticity can be applied, is distinctly diminished; these changes accordingly pave the way for dilatation of the vessels. It is impossible for any one correctly trained in the principles of physics to understand how Thoma should have been led into such an unfortunate misuse of the term. The



subject has been fully discussed by Thoma and myself within recent times.

On examining a section of an artery which has undergone sclerotic changes, it is found that the inner coat is considerably thickened throughout its whole circumference, but more especially in one spot, in the deeper layers of which there are frequently degenerative changes, shown by the presence of fatty globules, cholesterin crystals, and calcareous deposits. The internal elastic lamina is distinct in most instances. Sometimes, however, it may be observed to be broken up in some parts, while thickened in others. The middle coat is also increased in thickness, and under a high power may be seen to have more connective tissue amongst the muscle cells. There is likewise a thickening of the adventitia. These alterations are well shown in Fig. 193.

These changes are commonly diffused widely throughout the arterial system. It has been the habit to speak of diffuse sclerosis when the change involves great part of the arterial system, and to apply the term *nodose* to the form in which it is restricted to distinct patches. Such alterations may be found through the whole arterial system, and lead to narrowing and obliteration, or widening and rupture of vessels.

## ACUTE AORTITIS.

This process presents a twofold manner of occurrence, and the cases, therefore, fall into two categories. In one of these, the affection is found in the course of acute diseases, of which, therefore, it may be regarded as simply forming a part; in the other, it occurs as a primary lesion unaccompanied by any features denoting the presence of an acute general disease. It is of interest to note that in a very large number of cases of acute aortitis there has been some chronic change in the walls of the aorta before the acute process has begun. This is always the case as regards primary aortitis, but it is not invariably found in the primary form of the disease. Bureau is therefore perfectly correct in stating that there is probably no real primary acute aortitis,

and that the disease is always secondary either to a chronic aortitis or a general affection.

ETIOLOGY.—The acute general diseases, proved to have an intimate relation with the development of aortitis, are somewhat numerous. Like endocarditis, it may have its origin in rheumatism. Scarlet fever, measles, and smallpox have been, according to the researches of Landouzy and Siredey, Huchard, and Brouardel, followed by acute aortitis. Influenza has been observed in this connection by Fiessinger, while Huchard has observed acute aortitis following upon tubercular conditions. In such instances the affection is absolutely primary, and not to be engrafted upon a previous chronic change in the walls of the aorta.

Acute aortitis, arising independently of any general disease, seems, from the results of all observers, to be invariably associated with the previous existence of chronic disease. The affection is occasionally a sequel to acute endocarditis, and it cannot be doubted that, in some instances, the propagation is by direct infection. It is sometimes associated with pericarditis, pneumonia, and pleurisy; but whether the association is accidental, or an instance of cause and effect, cannot at present be determined. The affection has been found to follow upon renal disease in some instances. Pregnancy and parturition have been complicated by acute aortitis. The affection has been produced by over-fatigue and traumatism in the presence of impure blood. Boinet and Romary find infections and intoxications the sole exciting causes.

MORBID ANATOMY.—The aorta is almost invariably somewhat enlarged, and presents a fusiform aspect. On opening it there may be old-standing atheromatous lesions, but in the class of cases arising in the course of a general disease such appearances may be wanting. The surface is always rough and irregular, and the unevenness is due to patches, soft in consistence, and reddish or grayish in colour. These areas have a translucent aspect, so as to merit the term of gelatinous often applied to them. The patches have a tendency to run into each other, so as to form irregular outlines. Abrasion or ulceration of the surface is sometimes found, and it

will readily be understood that destructive changes of this kind may form a starting-point for the far-reaching effects of embolism. It sometimes happens that ecchymoses are present upon the aortic intima; these must be due to the rupture of some of the nutrient arteries. Not infrequently the whole of the internal tunic is deeply stained by the imbibition of the colouring matter of the blood, and the characteristic gelatinous patches may be almost of the colour of a blackberry from the same cause. Usually the middle and external coats of the aorta are somewhat thickened. They often contain a large number of newly formed blood vessels, and hæmorrhages take place into their tissues. In the more acute forms of the disease, the pericardium often shares in the acute process from direct extension. Microscopic examination of the affected patches shows that the internal tunic in its more superficial layers is invaded by a small cell infiltration, while large nucleated cells are also found. These are mingled with the ordinary stratified endothelial cells of the intima. The middle tunic is usually somewhat broken up by cell infiltration, and newly formed blood vessels ramify amongst the tissue elements. The same alterations may be found in the adventitia.

The affection has sometimes been found to set up changes in the cardiac plexus, and on rare occasions it seems to have been the starting-point of pleurisy. It is not infrequently associated with alterations in the aortic cusps, but these may be accidental coincidents, although facts in some cases seem to have proved that the aortitis has preceded the valvular lesion.

Two classes of acute aortitis have been described—one, characterised by the presence of vegetations, usually associated with a similar lesion of the aortic cusps, but occasionally, as in a case described by Boulay, found isolated; the other, resulting in suppuration, which has, within comparatively recent times, been very carefully investigated by Leudet.

**SYMPTOMS.**—It must be remarked that since acute aortitis so frequently arises in the course of another disease, its clinical features may be altogether overshadowed and completely masked by those of the affection which has preceded it. If it occurs in the course of an acute general disease, there may

already be some features of aortic implication, so that the aortitis causes but little additional disturbance; while in the case of pneumonia, or pleurisy, or, more particularly, endocarditis, there may be so many morbid appearances connected with the chest as to render the symptoms of aortitis almost imperceptible.

Amongst the general symptoms, it must be remarked that pyrexia is frequently absent; in fact, in most cases, it is never found. There is almost invariably a feeling of tightness in the chest, which very often occurs in paroxysms; or this feeling of weight and tightness may pass into a veritable angina pectoris. The pain in this case is distinguished from that of ordinary angina pectoris by the fact that in the latter there are periods of complete relief from all uneasiness, while in aortitis there is always a constant feeling of uneasiness and heaviness. The persistent feeling of uneasiness has been described by Leger as a burning sensation, occupying a position apparently in relation to the arch of the aorta, and passing to the back and down the spinal column. This has also been emphasized by Grainger Stewart. Tenderness, which has been more particularly investigated by Peter, is sometimes found in the first, second, and third intercostal spaces to the left of the sternum. Not infrequently complaints are made of dysphagia, which appear to be connected with the proximity of the aorta and the œsophagus. Vomiting, meteorism, and other symptoms of alimentary disturbance are sometimes described. Breathlessness may be persistent, but it more commonly occurs in the form of dyspnoea on exertion. Cough is a common symptom, and it is often attended by a considerable amount of expectoration.

Patients suffering from this affection are usually pale. They manifest, in short, the facies of aortic disease; no doubt, however, when there is an elevation of the temperature, there is a tendency to a higher tint upon the cheeks. On inspection of the neck, a considerable amount of throbbing may be found in the carotid arteries, and by Laboulbène and Faure an observation has been made, which is interesting in itself, whatever be the interpretation put upon it. This is, that the right subclavian artery pulsates more vigorously than the left.



The observers just mentioned believed the cause of this phenomenon to be that the innominate artery is less likely to be implicated than the left subclavian. Such a symptom is more apt to be caused by some previous chronic endarteritis.

The peripheral arteries may have healthy walls, or there may be some degeneration in them. Be that as it may, the radial artery is usually found to be only moderately filled, and its pressure is not high. The pulse wave is, as a rule, large and bounding, occasionally manifesting features like the pulse of Corrigan in regard to the duration of the wave. It may be regular or irregular according to attendant circumstances. It is not uncommon to find that the pulse wave is different in the two radial arteries; when this is the case, the left is usually smaller than the right, the reason for the difference being that some patch of disease interferes with the mouth of one or other of the arteries.

The præcordia may present no abnormal features on inspection. As, however, most cases of this affection follow a previous sclerotic change in the aorta, it is common to find that the apex beat is displaced somewhat outwards and downwards, while palpation reveals an increased intensity and prolonged duration of the impulse. It depends entirely, however, upon the stage at which the patient is seen, as towards the termination of a prolonged illness of this kind the heart usually fails, and the character of the impulse in such instances is like that described in dilatation. Percussion reveals, in the majority of cases, an increased area of cardiac dulness. The most important point elicited by this method, however, is an increased aortic dulness to the right of the sternum. On auscultation there may be nothing but an increase in the intensity of the aortic second sound. As a rule, however, the first sound in the aortic area is accompanied by a soft systolic murmur, followed by the accentuated second sound, and it is not uncommon to find that during the course of the disease a diastolic murmur is produced by implication of one or other of the aortic cusps, producing regurgitation.

The course of the affection is chiefly characterised by remissions. The disease is apt to be prolonged, and its

termination is so often fatal that it is scarcely an exaggeration to say that recovery is the exception. Death is brought about in certain cases suddenly by cardiac asystole, or by aortic rupture. When it occurs slowly and gradually, it is by cardiac failure, and is marked by the presence of œdema and other symptoms of a failing circulation.

DIAGNOSIS.—The recognition of acute aortitis is based upon the situation of the pain, and physical signs which denote an aortic affection. The means of differentiating between the pain of this affection and that present in angina pectoris, lies in the fact that in aortitis there are remissions but no real intermissions in the subjective sensations.

If septic phenomena, such as great fluctuations of temperature, and profuse perspirations, should be present, they will suggest the possible presence of suppurative aortitis; rapid loss of strength and tendency to cardiac failure will be found in such cases. If the appearances significant of embolism in any part should be present, they will inevitably suggest that some ulcerative process is taking place in the aorta. The sudden appearance of cyanosis with acute heart failure is to be regarded as almost pathognomonic of rupture of the aorta.

PROGNOSIS.—The outlook in this affection is always serious. It is somewhat less unfavourable in cases taking their origin in the course of some acute disease; it is absolutely hopeless in primary acute aortitis.

TREATMENT.—Absolute rest and appropriate food must be enjoined. The application of five or six leeches in the upper part of the sternal region has been found beneficial, especially when followed by the application of warmth, so as to favour bleeding. On the other hand, the use of the ice-bag, or of Leiter's tubes, has been of considerable service. The employment, further, of counter irritation has in certain instances been found to alleviate the symptoms. The pain is sometimes so severe as to imperatively demand a hypodermic injection of morphine. Of internal remedies, those which dilate the arterioles and lessen the blood pressure are chiefly serviceable; amongst them nitro-glycerin is certainly of most importance. The use of iodide of potassium will naturally suggest itself, and if there be much nervous disturbance,

bromide of potassium may be combined therewith. In cases characterised by a tendency to cardiac failure, the heart must be sedulously watched, and cardiac tonics administered if a condition of asystole should appear imminent. It need hardly be added that all these measures are rarely followed by recovery, except in a few of the cases having their origin in some acute general disease.

### CHRONIC AORTITIS.

The chronic forms of aortitis form a group of affections characterised by considerable variety, as well from the standpoint of pathological anatomy, as of clinical medicine. The lesions are of the deepest significance, inasmuch as they lead to so many consecutive changes.

ETIOLOGY.—The disease is usually part of general arterial sclerosis, and it therefore is produced by the factors which lead to that process. It is, in the first place, frequently observed in those who have a hereditary predisposition to degenerative processes in the arteries. It is essentially a senile disease, and is more common, therefore, in advanced years. The male sex is certainly more prone to it than the female. Certain disease tendencies produce a liability to chronic aortic changes. This remark is not intended to refer to hereditary tendencies, but to general acquired constitutional affections. Amongst these must be mentioned the diathesis which commonly bears the name arthritic, and there can be no doubt that the uric acid diathesis induces a liability to these affections. In one sense heredity plays an important part in this connection, seeing that the children of those who have an arthritic diathesis are certainly more prone to acquire it. There can be no doubt that specific infection has great influence in the production of such changes. Many observations have within recent years been made upon this subject by many authors, such as Welch, Lécorché and Talamon, Paul, Jaccoud and Döhle. On the other hand, Lancereaux throws some doubt upon the conclusions to which these observers have been led, and urges that impaludism is a very much more common cause. He forgets,

however, in making this statement, that the affection in this country, where it is so common, cannot often be produced by such a cause. Occasionally chronic aortitis is left behind by an acute process arising from an infection in the course of some general disease. This is, however, very much less frequent than the converse, and, as has been previously remarked, acute aortitis is much more likely to be engrafted upon a chronic process. Above all these factors, that which has been of most importance in the production of chronic aortitis is long continued overstrain of the aorta. The observations, commencing with Boerhaave, and culminating with Clifford Allbutt, have been mentioned, and need not in this connection be again referred to.

MORBID ANATOMY.—The lesions in chronic aortitis are sometimes entirely confined to the aorta, but it is much more common to find that the local alterations are only part of a general process widely spread throughout the whole arterial system. From comparison of the lesions in different individuals, as well as from the not infrequent coexistence in the same individual of different pathological appearances, it can easily be determined that the process goes through several distinct stages. The earliest stage consists in the development of patches characterised by opacity. These extend to some extent in all directions, and become raised above the surface, so as to form elevations; these may be flat—in which case each patch has the appearance of a plateau, or rounded—so as to present some resemblance to a button. These patches, which at first are somewhat grayish and gelatinous looking, become yellowish and doughy, with a great tendency to the development of hard masses, owing to the deposition of lime salts. The superficial portions of these elevated patches have a tendency towards erosion, which leads to the development of ulcers. These atheromatous ulcers present a somewhat rough floor of a yellowish colour, and the tissues which constitute them are obviously composed in part of fatty material. The aorta itself is almost invariably dilated; and in addition to such an increase in size, it is marked by the presence of smaller bulgings, which indicate the beginnings of aneurysmal changes. The aorta, when the condition is fully



developed, shows these various atheromatous alterations scattered widely throughout its extent, but they are more particularly seen close to the aortic orifice. One point of importance is that the lesions are very commonly seen at the origin of arterial branches. They are to be seen around the mouths of the coronary arteries, the great arteries of the neck and upper extremities, and even the intercostal arteries. The changes occasionally result in the formation of large calcareous plates, separated from each other by tissues more or less altered by the other atheromatous processes just mentioned.

On microscopic examination, the intima is found in the affected region to be sparsely invaded by round and fusiform cells enclosed in a fine network of fibrous tissue; according to Coats and Auld, characteristic branching cells, resembling those of the normal intima, are mingled with those which have been mentioned. In the deepest portions of the inner coat are masses composed of granular débris, mingled with oil globules, and crystals of cholesterin and margarin. These fatty changes take place in the cells of the intima in the first place. The middle and outer coats are always found to be vascularised as well as thickened, and they show an invasion by newly formed cell elements. These changes are undoubtedly compensatory.

This brief sketch of the morbid appearances would not be complete without some reference to the fact that chronic aortitis sometimes leads to rupture of the aorta, while, during the process of ulceration, embolism of distant organs may be the result of the separation of some of the necrosed tissue.

**SYMPTOMS.**—The clinical features presented by cases of aortitis do not form a uniform group. In some instances the affection may be perfectly latent until rupture of the diseased portion of the aorta brings about death in dramatic fashion; while, on the other hand, there may be appearances so distinctive in character as to be almost pathognomonic.

The common complaints are of uneasiness, weight, or constriction, along with palpitation or fluttering, as well as breathlessness, which may present the type of anhelation on exertion, or of paroxysmal cardiac asthma. Troublesome cough is not infrequently mentioned, and there may be

symptoms of giddiness or swimming connected with the nervous system. The pain is frequently of the true cardiac type—that is to say, it has the sternal position and the tendency to radiate along the great trunk lines already fully described. It is, moreover, often accompanied by the sensation as if the heart were going to stop, as well as by a consciousness of the near presence of death.

On careful examination of the circulatory system there is, in most instances, obvious implication of other arteries. The temporals, for example, are commonly rigid and tortuous, while the arteries of the extremities may give the feeling of twisted cords to the fingers of the observer. It is common to find that not only do the carotid and subclavian arteries pulsate excessively, but that in the jugular fossa the aorta itself may be found to give a well-marked impulse. The radial pulse, besides giving the sensation of hardness and tortuosity, is usually full, and manifests a high degree of blood pressure. The pulse wave is not usually large, although, from the sinuous or tortuous channel through which the wave of increased pressure travels, there may be a sensation as if the artery leaped from its bed. The pulse wave is sustained and dies gradually away with but little diastole. Such are the general features observed when there is a widespread arterial sclerosis. In the rarer cases, however, of localised aortitis, the radial pulse may manifest but few divergences from that which is normally found. A point of some importance is that the pulse may be strikingly different in the two radial arteries on account of the presence of a patch of atheroma at the mouth of one or other of them. The examination of the præcordia usually reveals the fact that the apex beat is further to the left and more diffuse than in health. On applying the hand, it is found to have the heaving sustained impulse of hypertrophy. If consecutive heart failure is undergoing development, however, the impulse may not present those characters, and may be short and sharp as in dilatation. The area of dulness is usually somewhat enlarged, more especially to the left. On percussion of the region to the right of the sternum from the third costal cartilage upwards, some dulness may be found considerably beyond the sternum in consequence of aortic dilatation. On

auscultation in the aortic area, the most significant feature is the character of the second sound. This has the distinctly musical character produced by a tap on a drum, whence Potain has aptly termed it *bruit de tabourka*, from its resemblance to the sound produced by the little Arab drum. The first sound in this region may be perfectly normal. Commonly, however, it is somewhat blurred, and, if aortic dilatation has followed upon aortitis, a systolic murmur may be present. In the mitral area the first sound is usually low and booming, but it may be high and sharp, according to circumstances. The pulmonary and tricuspid areas may call for no notice, but, if consecutive cardiac failure is present, there may be great accentuation of the pulmonary second sound, or the presence of a systolic tricuspid murmur may be determined.

It is not uncommon to find that œdema of the dependent parts is present in cases of old-standing, and, if this be the case, there is very apt to be implication of the bases of the lungs, shown by muffling of the percussion sound, prolonged expiration, and crepitations. Two accidents are apt to befall the lungs; one of these is rupture of one of the arterial branches, or thrombosis in it; the other is the rapid development of acute œdema. That the renal system is implicated is commonly proved by symptoms significant of cirrhosis. Affections of the nervous functions sometimes point to the existence of structural alterations produced by hæmorrhage, thrombosis, or embolism.

DIAGNOSIS.—As a general rule, the recognition of chronic aortitis, and its differentiation from other affections, present no difficulties. The characteristic physical signs connected with the aortic region and the absence of any proof of valvular disease, together with evidence of arterial sclerosis throughout the circulatory system, are sufficient to determine that the lesion is present. The chief difficulty arises when the chronic arterial change is purely local. It must be admitted that in many instances the affection may be absolutely latent, since, if no anginous symptoms are present, and there is no increased peripheral resistance from generalised sclerosis of the arteries, both symptoms and physical signs may be in default. If there be such subjective symptoms as have been referred to, along

with the characteristic second sound, the affection may be legitimately suspected.

PROGNOSIS.—Once established, chronic aórtitis never disappears. In this sense the prognosis of the affection is accordingly grave. As it is, nevertheless, possible to retard the disease processes by appropriate management, the outlook is very largely dependent upon the attendant circumstances. If the condition of the general nutrition and energy, as well as the state of the heart and circulation, should be fairly satisfactory, the prognosis need not be very grave.

TREATMENT.—The management of chronic aortitis is practically that of arterial sclerosis. Abundance of rest, sufficiency of fresh air, diet mostly composed of white meat, vegetable substances and milk, with free diluents not containing alcohol—such are the most useful lines of treatment. Of medicinal substances, those which act upon the blood vessels are of most service, and amongst them iodide of potassium is the most valuable. In many instances the action of the iodide is increased by combining it with arsenic. When cardiac failure steps in, it must be treated on general principles, and any paroxysms of angina pectoris will require the special remedies applicable to that symptom.

### ANEURYSM OF THE AORTA.

So far as is known, the earliest notice of aneurysm of the aorta is that of Fernel, but the first diagnosis during life is due to Vesalius. Baillon observed abdominal aneurysm, while Albertini and Valsalva not only extended the existing knowledge of the clinical features of the disease, but introduced some of the therapeutic methods still in vogue. The great work of Lancisi remains to this day a wonderful storehouse of facts regarding the disease, and the pages of Morgagni teem with most interesting observations on it. The name of Scarpa will ever be indissolubly associated with aneurysm, in consequence of his work on it. Corvisart and Laennec were the pioneers in the modern diagnosis of the affection, while its morbid anatomy received its first real impetus from Hodgson.



In so far as is possible, the names of those who have made advances in the study of the disease will be remembered in the following pages.

ETIOLOGY. — Numerous causes predispose to aneurysm. Many of these have already been mentioned in connection with endarteritis, and but little remark is necessary in reference to them. Some of these factors, however, have a more marked influence in the production of aneurysm than of endarteritis, and their relations are, therefore, of more importance in this connection.

There can be no question of the much greater tendency towards aneurysm of the aorta manifested by men as compared with women; since the observations of Hodgson and Bizot this fact has been matter of universal recognition. The former of these observers found that of 63 cases, 56 occurred in males, and 7 in females; while the latter in 189 instances found 171 in men as against 18 in women. Browne has recently in his analysis of 173 cases found 153 in men and 20 in women. That the influence of sex is brought about by the different conditions under which average men and women live, is well shown by an observation of Coats, who points out that, while aortic aneurysm occurs in a preponderating manner in men, cerebral aneurysm is found almost equally in both sexes.

Age is found to show an interesting relation in regard to the incidence of aneurysm of the aorta. The most important series of statistics dealing with this question is that of Crisp, who found that out of 505 cases of different aneurysms, 198 occurred between thirty and forty, and 129 between forty and fifty. Of 163 cases Browne found 67 between the ages of thirty-five and forty-five, and 46 between forty-five and fifty-five. Similar statistics, although on a smaller scale, have been collected by other authors, and from these the conclusion is unavoidable that aneurysm is more frequent in the fourth decade. Coats remarks with great justice: "Aneurysms coincide with the time of life when the period of greatest bodily vigour overlaps the beginnings of the period of occurrence of atheroma." Endarteritis shows itself at a somewhat later period.

Race appears to have some influence, and there can be no doubt that the inhabitants of the British Isles are more liable

to the affection than other nations. This is almost certainly due to the much greater stress under which many of them have to live on account of special occupations. Among predisposing causes, certain diathetic conditions are certainly of importance. The uric acid diathesis in its various manifestations must here be accorded an important place, and it is probable that rheumatism also must be allowed some influence of a similar kind. Specific invasion, as asserted by Welch, probably plays a somewhat important part in the production of conditions leading to aneurysm. A very great amount of discussion has been devoted to the subject; Douglas Powell and Gull, for example, have been sceptical as to the connection between syphilis and aneurysm, while Jaccoud has supported the views of Welch. The coexistence of infection and aneurysm cannot for a moment be doubted, and it seems to me that the demands for a special type of lesion underlying the development of aneurysm, which have been put forth by the opponents of Welch's view, may just as well be made against the view that any dyscrasia exercises an influence in the production of the disease.

The abuse of alcohol and other toxic agents may undoubtedly predispose to aneurysm by leading to degenerative processes, and the same observation may be made in regard to all lesions of the arterial walls by which their elasticity is diminished.

The exciting causes of aneurysm, at least as regards the thoracic aorta, are almost entirely based on the conditions of blood pressure. It must be allowed that the abdominal aorta, like the arteries of the limbs, may be the seat of the disease in consequence of traumatism, but it must be rarely indeed that either direct or indirect violence can produce thoracic aneurysm. With the exception of this possibility, the determining factors in the production of aneurysm are centred in an increase of blood pressure. It is through this agency that age and sex have their chief influence in giving rise to aneurysm. Severe exertion of any kind produces an elevation of the blood pressure, and it is found that all occupations which cause great fluctuations in the blood pressure are especially prone to aneurysm.

It should be added that amongst the determining causes it is possible that direct injury from the impact of emboli upon the arterial walls must be included. This was suggested originally by Church, and the idea has been further elaborated by Ponfick. Although it must be admitted that such a cause may be operative in regard to aneurysm arising from the smaller arteries, it certainly seems in the highest degree unlikely that such an etiological factor obtains in the case of the aorta.

Another consideration as regards the etiology of aneurysm also requires notice. As has long been known, a change in the walls of the aorta is sometimes found to follow the lines of distribution of the nutrient vessels. By Martin, the conception of an obliterative endarteritis affecting the vasa vasorum has been regarded as the starting-point of the changes in the wall of the aorta which precede the development of the disease.

MORBID ANATOMY. — There can be no doubt of the relations existing between sclerosis and aneurysm. The connection is absolutely proved by the presence of atheromatous changes in every case of aneurysm of the aorta. The relations of the two lesions, which have been recently the subject of exhaustive investigations by Coats and Auld, and by Hollis, can be studied most satisfactorily in such cases as manifest the commencement of the aneurysmal change in small depressions or patches on the inner surface of the aorta. Even with the naked eye it may be determined that the hollow coincides with a patch of disease, and on microscopic examination of a section of such a depression it is found that the media has undergone atrophy. The middle coat becomes gradually thinner on passing from the periphery of the depression towards its centre, and at this latter point it is often almost entirely absent. There can be no doubt that this change in the middle coat is produced by a previous alteration in the intima, which by invading the media leads to atrophic processes with or without definite reaction phenomena. When the latter are present there may be cellular proliferation and vascularisation, whereby attempts at compensation are produced. These different processes are con-

tinued after the production of the aneurysm, and great part of its wall is composed of connective tissue formed by such reaction processes.

The microscopic appearances are shown in the accompanying illustration (Fig. 194).

The appearances presented by aneurysm have been subjected to a process of analytic classification of the most searching



FIG. 194.—Changes in the aortic walls leading to aneurysm. The intima (*a*) is greatly thickened; the media (*b*) shows great atrophy, and, at one part, almost total disappearance; the adventitia (*c*) has considerable increase, and, in its engorged and dilated blood vessels, furnishes evidence of great vascularisation.

kind. It is not my intention to follow in the suit of such unnecessary, if ingenious, subdivision. It is quite sufficient to arrange aneurysms in three great groups:—true, dissecting, and false. By true is meant the condition in which the arterial tunics in whole or in part form the walls of the aneurysm; the term dissecting is applied to conditions in which the blood has passed between the coats, amongst which it forces its way so as to separate them from each other; while the variety termed false cannot be regarded as in reality an aneurysm at all, seeing that it is produced by a rupture of



the arterial walls, leading to the formation of what is simply hæmatoma. The first-named variety is that to which alone full consideration will be given in the following pages, although a word may be allowed in regard to some of the appearances produced by dissecting aneurysm.

True aneurysm gives rise to morbid appearances usually classed under two heads, but these can be regarded as the two extremes of a series united by intermediate forms.

Dilatation of the aorta, or fusiform aneurysm, is most commonly found in the ascending and transverse portions of the arch. It is much less commonly found in the descending aorta. As a rule the dilatation does not only affect the aorta, but also the arterial branches which arise from the part affected. In dilatation of the ascending and transverse portions of the arch, it is extremely common to find that the innominate artery and even its branches have undergone a similar change. In this respect simple fusiform dilatation is very different from sacculated aneurysm, which is rarely linked with dilatation of any of the arterial branches. The dilatation is, for the most part, almost uniform. In the abdominal aorta, for example, if there be any fusiform enlargement, it is almost invariably equal in every direction. This, however, is not the case when the dilatation affects any portion which is curved, as in the case of the arch of the aorta; it then is found to be much wider on the side of the larger curvature, no doubt from the direction of the stream of blood upon it. The walls of the aorta are always thicker than in health. This is due to a change in all the coats. The intima shows some of the alterations described under chronic aortitis; the middle tunic is almost invariably diminished in thickness; the adventitious coat is always augmented to a considerable extent by the result of reaction processes. There is much less tendency towards coagulation of blood in a fusiform aneurysm, and there is accordingly much less deposition of fibrinous layers on the inner aspect of the cavity.

Sacculated aneurysm presents immense varieties in size. Indeed, it may be held, without straining the facts, that its dimensions may vary from a bulging in the wall of the aorta not exceeding the size of a large pea

to an enormous sac as large as a man's head. The walls of an aneurysm are always formed, in part at least, by the arterial coats, but the extent to which they participate in the formation of the sac is subject to considerable variations. The intima invariably has some share in the formation of the sac, and it can be traced from the edge of the opening for some distance along the inner lining. In the smaller-sized aneurysms it may be continued throughout almost the whole of the sac, but in those of larger size it, for the most part, is only carried along the inner aspect for a comparatively short distance. The middle tunic fades away close to, or even outside of the commencement of the sac. This is what might be expected from the fact of its undergoing a previous atrophic change. The adventitious coat, which has already been shown to be relatively thinner in the aorta than in other arteries, becomes greatly increased in aneurysm by reaction processes taking place in it. It also becomes very much more vascular, and, as will be mentioned below, has a tendency to adhere to neighbouring tissues.

The opening between the lumen of the aorta and the cavity of the aneurysm is subject to great variations in size. In some cases it does not exceed two or three lines, while in others it may reach two or three inches. The cavity may present one or more chambers, since it is sometimes broken up by partitions, formed by organised deposits. The inner aspect usually presents a somewhat rough surface. A considerable quantity of newly formed tissue is disposed in a laminated manner upon the inner aspect, and the layers so deposited, along with organised blood clots, give rise to irregularities. The contents partly consist in the blood in a fluid state, but along with this there are usually some coagula, which may be more or less recently formed. The walls of the aorta elsewhere are usually found to be altered in appearance, the various changes which have been described under the head of chronic aortitis being present. In a few cases, with the exception of the portion of the aorta immediately adjacent to the aneurysm, the walls may be found wonderfully healthy. This, however, is decidedly rare. The intima is very frequently implicated by sclerotic or atheromatous changes at the origin of the arterial branches. In a

very large proportion of the cases of aortic aneurysm there is some affection of the semilunar cusps. Most frequently such changes are of the nature of sclerosis, obviously having origin in factors analogous to, if not identical with, these which have given rise to the formation of the aneurysm. The state of the heart has been much discussed. Reference to this subject has been made in the consideration of cardiac hypertrophy, and all that requires to be mentioned in this connection is the fact, more particularly emphasised by Stokes and Axel Key, that hypertrophy of the heart is by no means an invariable or necessary sequel to aneurysm. Browne shows that in only 72 out of 173 cases of aortic aneurysm did hypertrophy occur.



FIG. 195.—Sacculated aneurysm arising within the sinus of Valsalva and involving the interventricular septum.

Aneurysm, by its presence, effects considerable changes in adjacent textures. In the first place, it invariably gives rise to more or less displacement. In aneurysm of the thoracic aorta, the position of the heart itself may be modified, and the lungs, the trachea, the bronchi, the œsophagus, and the various structures within the mediastina may undergo considerable displacement. Such alterations in position are almost as well marked in the case of aneurysm of the abdominal aorta. By pressure upon neighbouring structures, moreover, tissue changes are also produced. Adhesions are formed between the wall of the aneurysm and the textures upon which it presses, while erosion of certain of these tissues is common. There is



further a great tendency to obliteration of hollow viscera, and alteration of the texture of such organs as are compressed. Total occlusion of a bronchus with resulting collapse of the corresponding lung has often been observed, while it is a matter of everyday experience to find that by compression the lung tissue may undergo such changes as to render it remarkably like the spleen in its naked eye appearance. In rarer cases the heart walls may be invaded, as in the case kindly placed at my disposal by Dr. Harvey Littlejohn, from which the preceding illustration, Fig. 195, was taken. In it an aneurysm arose in a sinus of Valsalva and involved the interventricular septum.

It occasionally happens that an aneurysm undergoes spontaneous resolution, so to speak, either because the sac becomes entirely obliterated by deposits from the blood, or by closure of the orifice leading to it. Such results are extremely rare in the case of aortic aneurysms, and they therefore present a marked contrast to the events which are commonly found in the case of aneurysm of the peripheral arteries, in which, by pressure upon the artery from which the sac projects, it may be obliterated, and the sac thereupon becomes filled with organised blood clot. A very much more common event is rupture of the wall of the aneurysm, and escape of its contents into some neighbouring part. Browne found that this occurred in 84 of the 173 cases which he analysed.

Dissecting aneurysm is produced by rupture of the internal tunic, permitting the presence of blood between it and the middle coat, or between the layers of the middle coat. In either of these positions it is apt to find its way for some distance along the vessel, and may establish a communication again with the lumen of the vessel, so that the false passage forms the main arterial channel. On the other hand, it occasionally, by penetrating the outer coat, causes rupture of the aorta. Kelynack has recently published the history of this affection. A beautiful example of this affection, kindly presented to me by Dr. Harvey Littlejohn, is shown in Fig. 196.

SYMPTOMS.—The symptoms of which complaints are made by those who suffer from aortic aneurysm are extremely variable, but the most common are undoubtedly pain, palpitation, and



breathlessness. It is by some authors the custom to divide the symptoms of the affection into direct and indirect ; while



FIG. 196.—Dissecting aneurysm of the ascending aorta. The walls of the aneurysm are kept apart by a pencil stem. The aorta shows great atheroma.

by others it has rather been the custom to separately study the rational symptoms and physical signs. It is certainly easier to marshal the facts if they are systematically analysed

according to the systems affected. The symptoms must, however, be classed under the heads of general and local.

#### GENERAL SYMPTOMS.

*Circulatory Symptoms.*—It frequently happens that subjective sensations of the kind observed in cardiac disease make their appearance. Anginous attacks, radiating to one or other shoulder and arm, according to circumstances, are tolerably frequent. Along with such sensations there are not uncommonly feelings of fluttering or throbbing, which call attention to the fact of some circulatory disturbance.

Inspection of the patient may show the pallor which is common in aortic disease, and the arteries of the neck often pulsate with excessive vigour, while a capillary pulse may be observed along with these phenomena, if there be, as is so commonly the case, aortic incompetence. The apex beat may be observed in its ordinary position, but it is common to find it displaced in different directions. Some modification in the form of the chest may be present. Sometimes this occurs in the form of a diffuse bulging, but at other times it constitutes a distinct tumour. In either case the part thus prominent presents the appearance of pulsation.

On palpation, the apex of the heart may be found to give the characteristic impulse of cardiac hypertrophy; more commonly, however, it is unchanged or manifests unmistakable evidences of dilatation. On applying the hand over the swelling, if such be present, the fact of pulsation is amply confirmed, and it can usually be determined that it is of an expansile character, while in a certain proportion of cases it is accompanied by a distinct thrill. The sense of touch often reveals the fact that the pulsation of the tumour follows that of the apex of the heart by an appreciable, if short, interval, which, speaking generally, can rarely be ascertained by the unaided sense of sight. By attaching two flags to the chest with wax, one over the apex and another over the swelling, the fact may be better seen. Tracings obtained from aneurysms show considerable divergences from the usual arterial curve. A sphygmogram from a dilatation of the innominate and carotid arteries is

given below (Fig. 197), and it shows a swift ascent, a flattened summit, and a rapid descent. A curve of a somewhat similar character is seen in Fig. 198, which is a tracing taken from an aneurysm of the ascending aorta. In both the double summit is distinct, and it is the graphic representation of the twofold impulse often felt.

A change in the position of the heart may be determined

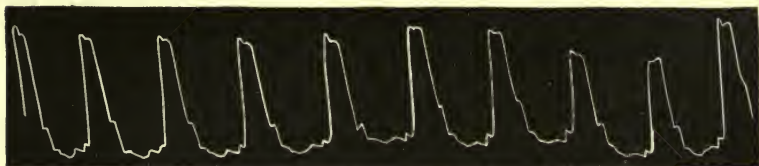


FIG. 197.—Tracing from fusiform aneurysm of the innominate and common carotid arteries, obtained by means of the direct cardiograph.

by percussion, and sometimes also an alteration in its size, in the direction of enlargement. Over the swelling or tumour the percussion sound may be absolutely dull, and the total extent occupied by the aneurysm may be ascertained with considerable exactitude by this means; percussion occasionally also reveals the presence of dulness in situations where inspection and palpation fail to detect anything abnormal.

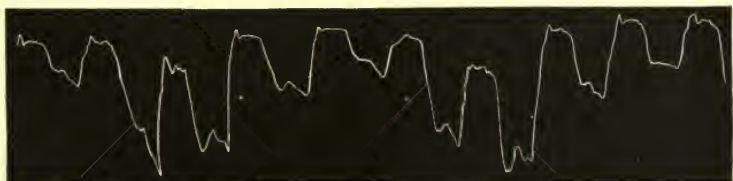


FIG. 198.—Tracing from sacculated aneurysm of the ascending aorta, obtained by means of the direct cardiograph.

This is especially the case in aneurysm of the descending aorta above the diaphragm. In cases of this kind, percussion may furnish most useful evidence, taken in association with the results of other methods of examination.

Over the seat of the lesion either the normal arterial sounds or abnormal murmurs may be heard. When pure sounds are audible in the case of aneurysms situated near the heart, the sounds are double, that is to say, the two sounds heard over

any large artery are present. These sounds were originally studied by Stokes, Bellingham, and Lyons, and a very considerable amount of discussion took place as to their production. All this may now be regarded as matter only of historic interest, and the sounds must be regarded as propagated from the heart, just as is the case in the arterial sounds, which have been discussed previously. In many instances these sounds are not present, one or both being replaced by murmurs. By far the most frequent abnormal sound is a systolic murmur, the origin of which is probably by no means always the same. It is possible that it may be produced by a current passing into the aneurysm and causing vibrations; it is, however, more probable that the murmur is produced by eddies within the sac, which have their starting-point in the current as it passes the aperture, or in the small quantity of blood which enters the sac. There is another mode of production which may reasonably be expected to occur frequently, that is, the pressure of the sac upon the aorta, which may cause an alteration in the lumen, and thus furnish such conditions as are well known to produce a murmur. In many instances some obstruction of the aortic orifice is associated with an aneurysm of the aorta, and a systolic murmur in such cases, having its origin at the orifice, is simply propagated by means of the sac. This may be held to be the cause of the systolic murmur in a good many cases. A diastolic murmur is not infrequently heard over the aneurysm, and a considerable amount of discussion has arisen with regard to its method of causation. It may be said, without fear of contradiction, that in almost every case presenting a diastolic murmur there is coincident incompetence of the aortic cusps. Quincke usually has the credit of having called attention to this undoubted fact, but, long before the appearance of any work of his upon the subject, Corrigan had described it. Within my knowledge, there are only four well-authenticated cases of sacculated aneurysm in which a diastolic murmur was present along with a healthy condition of the aortic cusps—the interesting case described by Balfour, who himself confesses his inability either to explain or understand the production of the murmur, and those recorded by Legg, Bryant, and Pringle. That it is produced



by a reflux during cardiac diastole out of the sac into the artery may well be doubted, not only since the amount of blood which passes out of the sac during diastole must be comparatively small, but because on the cessation of the cardiac systole with perfectly competent aortic cusps, the diminution of blood pressure which ensues occurs as a somewhat gradual process, and cannot produce the necessary conditions for the origin of a diastolic murmur. It may be, as Quinke suggests, that aortic regurgitation is sometimes found as a purely functional disturbance without any structural alterations; but this, to say the least of it, must at present only be regarded as an unproven hypothesis.

A distinct diastolic murmur was described by Marey as occurring in a case of dissecting aneurysm communicating with the aortic channel at one of the sinuses of Valsalva, apparently without aortic incompetence. The explanation of such a condition is certainly difficult, but it is quite possible that, after the termination of the systole, the blood in the intermural space might, while the aortic pressure was falling from its continuous peripheral outflow, escape into the intramural channel with sufficient force to generate a murmur which would necessarily be late diastolic.

Auscultation of the heart very commonly gives evidence of aortic murmurs, denoting obstruction, regurgitation, or both, and it is an interesting point that such murmurs are occasionally less distinct in the conventional aortic area than over the aneurysm. The same may be said of the sounds heard over the heart, which are sometimes less distinct than over the sac. Auscultation furnishes proof in some instances of mitral obstruction or incompetence, but by no means so commonly as is the case with the aortic orifice.

The arteries, where they are accessible, are usually hard and tortuous, but are sometimes, on the contrary, perfectly yielding and elastic. The radial pulse may manifest considerable variety in its appearances. The walls are usually rigid, but the fulness of the vessel presents no uniform condition. In some instances the contents are certainly much above the normal, but in others the vessel may be much less full than in health. The pressure also is subject to similar variations.

The rate, as a rule, is rather lower than the healthy average. With regard to rhythm, the pulsation is, except in cases of approaching cardiac failure, perfectly regular. The individual pulsations have considerable variations in different cases. Sometimes the wave is small, tardy, and sustained; at other times, and this is perhaps more commonly the case, the wave is large, swift, and evanescent. The differences in regard to the pulse depend upon two considerations: in the first place, whether there be much general arterial sclerosis, and in the second place, whether aortic incompetence be present or not.

The pulsation in the different arteries of the body occasionally reveals facts of considerable interest, and even of diagnostic importance. When the radial pulse of the two arms is compared, it may be found to present differences both in time of occurrence and form of curve. It has long been held, as indeed has been stated by Quincke, that the difference in size and time is due to the fact that the sac acts as a sort of reservoir. This, however, has long seemed wanting in conclusiveness as an explanation, and the idea may be said to have received its deathblow at the hands of von Ziemssen, who has shown that such a difference is produced either by direct pressure upon the mouth of the branch in which the retardation and diminution are to be found, or that some patch of atheroma has caused occlusion at that part. Sphygmographic tracings, showing such changes as have been referred to, will be found in the sequel.

A considerable amount of venous stasis and cyanosis may be found in consequence of pressure upon one of the great venous trunks, and consequent interference with the return of the blood. Amongst other phenomena, a direct venous pulse has been described, but this is never the result of the affection itself, and is only found associated with it when aortic incompetence is also present. This statement does not apply to cases of aneurysm in which, from some pyrexia, a direct venous pulse has been produced through dilatation of the arterioles.

Pulsation of the arteries in the retina has been described by Becker, and in one instance of aneurysm of the arch the pulsation was found to be more distinct on the left side than on the other.

*Alimentary Symptoms.*—The digestive system may be implicated directly or indirectly. By pressure upon the œsophagus some difficulty in swallowing is often produced, as has been known since the observations of Morgagni. Besides this, there may be spasm of the pharynx and reflex dysphagia. All these symptoms connected with the œsophagus are found more particularly when the lesion affects the transverse portion of the arch and the descending thoracic aorta, but Walshe mentions such a result of abdominal aneurysm. In consequence of the yielding character of the abdominal contents, aneurysm of the aorta below the diaphragm does not produce many direct symptoms in connection with the digestive organs, yet it must be remembered that while direct implication is by no means common, a good deal of reflex disturbance may be produced in consequence of interference with some of the visceral nerves. There may, therefore, be symptoms of excessive or diminished nervous activity connected with the abdominal organs.

*Hæmopoietic Symptoms.*—The hæmopoietic system does not commonly show many changes, but there are two which may be referred to. One of these, originally observed by Laennec, is pressure upon the thoracic duct by aneurysm of the descending aorta. When this takes place, it produces considerable interference with the blood-forming apparatus, and emaciation may be the result, associated with enormous distension of the thoracic duct. Another rare occurrence has been described—the production of pigmentation of the skin, in every way resembling Addison's Disease, from pressure upon the semilunar ganglia, in the total absence of any change in the suprarenal capsules.

*Respiratory Symptoms.*—The respiratory functions are often interfered with, and breathlessness may have an immediate or remote origin. By direct pressure upon the lung itself, the aerating space may be considerably reduced, while by pressure upon one of the bronchi there may be collapse of an entire lung; in both these instances there is therefore a direct cause for dyspnœa. In certain rare instances, dyspnœa has also been produced in a somewhat direct manner by pressure upon, and occlusion of, the pulmonary artery, while pressure upon the pulmonary veins has likewise, through the production of stasis, also led to breathlessness. In addition to these more



or less direct methods of production of breathlessness, the symptom may have its origin in interference with the vagus nerve, in which case a reflex dyspnœa results. Cough may be caused by pressure upon the trachea in the case of aneurysm of the transverse portion of the aorta. In such a case, the cough is of a peculiarly harsh, strident description, so much so as to merit comparison to the cough of a gander, as has been suggested by Wyllie. When there is pressure by an aneurysm of the descending aorta upon the left bronchus, a persistent cough is also often present, but in this case its character is much less peculiar than when the trachea itself is compressed. Cough, like breathlessness, may also be of reflex origin, and when there is implication of the vagus nerve, or its branches, such an indirect cough is often found. Hæmoptysis may attend the cough. This may be the result of congestion from pressure, but the possibility must ever be borne in mind that it may have its origin in weeping from the aneurysm into some part of the respiratory tract. Suffocative symptoms cannot be regarded as of frequent occurrence, but they nevertheless present themselves occasionally as the result of interference with the glottis through pressure upon the laryngeal nerves. This subject will again be referred to in considering the nervous effects of the disease. Wheezing noises, having their origin in the respiratory tubes, are of very frequent occurrence, and sometimes present the characters of intense stridor. Many cases of this kind, in which aneurysm was ascertained to be the cause, may be found in the pages of Morgagni.

An alteration in the form of the chest is sometimes found in consequence of recession of some part of the chest. When this occurs, it is usually the result of pressure upon one of the larger bronchial tubes, if not on a bronchus. In cases of this kind there may further be seen less movement upon the affected side. Occasionally diminished movement of the left half of the diaphragm may be found in consequence of interference with the phrenic nerve, as will be afterwards more fully mentioned. One important symptom connected with the respiratory organs is observed on inspection, that is, the symptom of tracheal tugging, first observed by Oliver, MacDonnell, and Ewart. This is observed when the



patient bends the head backwards, so as to put the tissues in front of the neck on the stretch. In this position the trachea will be found to be dragged downwards with each contraction of the heart, and if the finger be placed between the thyroid and cricoid cartilages, and the former be gently pulled upwards, there will be a distinct sensation of dragging with each cardiac revolution. If not absolutely pathognomonic of aneurysm of the transverse portion of the arch of the aorta, this symptom is, at any rate, seldom observed when it is situated elsewhere. On palpation, if well-marked stridor be present, the vibrations may be distinctly felt, and if there be collapse of any part of the lung from pressure, increased vocal fremitus may be elicited. Dulness may be found over any region which may have undergone consolidation from compression or collapse in consequence of obliteration of the respiratory channel. There may be an increase or decrease of the respiratory murmur. In those cases marked by consolidation, in which the affected area is in connection with any of the larger air-tubes, the breathing may be of extremely harsh vesicular character, or may even pass into the bronchial type. On the other hand, if obliteration of a bronchus has taken place, there may be an almost total loss of the breath sound, and it need hardly be added that the voice undergoes analogous increase or diminution under similar circumstances. Various accompaniments of the breath sounds may be present according to the conditions which have been induced. When there is much interference with the circulatory conditions in the lungs, there may be some moist sounds, but entirely surpassing these is the presence of the stridor already mentioned. Loud as it is to the bystander, it is still more distinct and characteristic on auscultation, and it may present wide gradations in pitch and harshness. Over and above all these respiratory symptoms, there may occasionally be the development of such characteristic *foetor* and *cachexia* as, along with the physical signs of lung implication, to prove the development of pulmonary gangrene.

*Cutaneous Symptoms.*—The skin, as has already been mentioned, may show cyanosis, either locally or generally, and, along with the subcutaneous tissues, it may exhibit *œdema* of the dependent parts from consecutive cardiac failure, or of

some restricted area corresponding to one of the great venous trunks. One very interesting observation, originally made by Gairdner, which occasionally presents itself, is a unilateral, or, indeed, strictly localised perspiration from interference with the sympathetic nerves. The skin has also been observed by Walshe to have localised areas of temperature higher than that of the rest of the body.

*Urinary Symptoms.*—The urinary system may be said to be wonderfully exempt from interference, save in the few cases showing albuminuria from pressure on the renal veins.

*Generative Symptoms.*—The generative apparatus in the male may undergo structural changes; thus Walshe mentions atrophy of the testicle from pressure on the spermatic artery. It might theoretically be expected that in women the existence of aneurysm low down in the abdomen might directly affect the uterus and ovaries, but this is, so far as is known to me, unrecorded.

*Nervous Symptoms.*—The nervous effects of aneurysm are multifarious. Many different sensory symptoms have, since Morgagni's original descriptions, been well known. Sensations of weight, of tightness, of soreness, or of pain are felt in many different degrees. Some of these pains are due to direct pressure upon nerves of ordinary sensibility, as, for instance, when a thoracic aneurysm interferes with an intercostal nerve. Most of them, however, are of the nature of referred sensations, and must be placed in the same category as the cardiac pain already fully discussed. In a large proportion of cases of aneurysm of the arch of the aorta, angina pectoris, in its most pronounced and unmistakable forms, is present. It is probable that interference with afferent nerves is the cause of many spasmodic symptoms, such as a spasm of the glottis, or of the pharynx, from interference with the vagus nerve; but it must not be overlooked that the former may possibly be an early symptom of interference with the motor nerve of the larynx. The recognition of paralysis of the laryngeal muscles, from pressure upon the vagus or recurrent laryngeal nerve, is often ascribed to Traube, but was previously observed and explained by Stokes. The left vocal cord is that which is usually implicated, seeing that the recurrent laryngeal nerve of that side passes

round the transverse portion of the arch of the aorta. In the case of dilatation of the ascending portion of the arch, extending into the innominate and subclavian arteries, a similar result occurs on the right side. Both cords have been affected from unilateral pressure, as seen by Bäumler and Johnson. Semon has recently shown that the abductors suffer first. When the larynx is examined with the laryngoscope, the cord of the affected side is found to occupy the position known as the cadaveric. The unilateral paralysis of the diaphragm, as originally noticed by Burns, may result from aneurysm pressing upon the phrenic nerve. This occurs on the left side when the descending aorta is the seat of the lesion. Changes in the size of the pupil are of common occurrence, as the result of pressure upon the sympathetic nerve anywhere above the lower extremity of the cilio-spinal portion of the gangliated cord. This interesting phenomenon was first brought into prominence by Gairdner. Pupil changes, from pressure by other lesions upon the sympathetic nerve, had been previously described as mentioned by Reid, and Walshe noted, but did not explain, them in aneurysm. It was nevertheless Gairdner who elevated the symptom into an important sign of the disease. The observations of Ogle and Argyll Robertson on this subject are also of real interest. The appearances are by no means uniform. In an early stage of pressure upon the sympathetic nerve, the pupil on the affected side is often widely dilated, and its size varies, not merely from day to day, but from hour to hour, while its condition becomes much less marked in the case of an aneurysm which is undergoing improvement; at a later stage, when the sympathetic nerve has evidently been destroyed by pressure or adhesions, the pupil becomes much contracted from the unbalanced action of the circular fibres. Such symptoms may be found in thoracic aneurysm arising from almost any part of the aorta, while similar effects have been described by Seatoun Reid as the result of aneurysm of the abdominal aorta. Amongst motor symptoms, it must not be forgotten that paraplegia may be produced by erosion of the vertebral column and spinal cord, but is decidedly rare.

*Osseous Symptoms.*—The bones frequently undergo destruc-

tive changes in consequence of pressure. The sternum and ribs in front of the thorax, and the spine and ribs behind, are all liable to undergo erosion and necrosis from pressure upon them, and consequent interference with their nutrition. The result of such changes is to interfere very greatly, not only with the form, but with the movements of the chest.

#### LOCAL SYMPTOMS.

It is advisable to consider the symptoms of aneurysm of the aorta in different situations, in order to bring together the clinical features characteristic of its various positions.

Aneurysm of the *ascending portion of the arch of the aorta* gives rise, in the great majority of cases, to a distinct pulsating tumour, situated to the right of the sternum in the second and third intercostal spaces. Some rare instances, in which the sac projects backwards and inwards from the lesser curvature of the aorta, do not produce the localised pulsating swelling. The apex beat is usually displaced somewhat outwards and downwards, and it is easy to determine that there are two points of maximum impulse upon the chest. On applying the hand over the tumour, a double impulse is usually felt, the first of the two impacts being occasionally attended by a thrill. Percussion affords the means of defining more accurately the exact position and size of the aneurysm. On auscultation, there may be the two sounds previously described, or more commonly a systolic murmur followed by a short sharp sound, unless in those cases associated with incompetence of the aortic valve, when systolic and diastolic murmurs are both heard over the sac. Such are the most frequent direct appearances found in aneurysm in this position. Some other features may be present. On auscultation, for instance, pericardial friction is occasionally found, connected, as was seen in dealing with diseases of the pericardium, with the pressure of the aneurysm. The right pulse may be smaller in size, and even slightly later in time than that of the left, if the aneurysm causes interference with the mouth of the innominate artery. Localised cyanosis or cedema of the right side of the face and neck, as well as the right arm, may be present. There is



frequently intense pain darting down the right arm, and it is not uncommon to find that the muscles of that limb are decidedly weak, or even atrophic. In some instances there may be aphonia, from paralysis of the right vocal cord, if the disease implicates the innominate and subclavian arteries, as is sometimes, but rarely, the case. Contraction of the right pupil is much more common than any affection of the larynx, since the aneurysm is much more likely to interfere with the sympathetic than with the right recurrent laryngeal nerve. Breathlessness, cough, and expectoration, not of diagnostic importance in themselves, frequently lead to a careful examination of the lungs, when it is common to find a considerable amount of retraction of the right lung.

Aneurysm of this portion of the arch very commonly ruptures into the pericardial sac, but it also not infrequently bursts into the right pleural cavity, or by means of adhesions it may become attached to, and finally rupture into the right lung. Sometimes it opens into the superior vena cava. In other cases it brings about a fatal termination by rupturing externally through the thoracic walls.

Aneurysm of the *transverse portion of the arch of the aorta* gives rise to a diffuse heaving impulse in the upper sternal region, and episternal notch. In some cases, which are, however, by no means common, the aneurysm by erosion produces a distinct swelling, projecting through some portion of the upper sternal or upper costal region. The impulse which is felt on laying the hand over the præcordia cannot be so distinctly made out to be double. This may, however, sometimes be ascertained with sufficient distinctness. On the other hand, a thrill is occasionally, but not at all commonly, found. A difference in the character of the two radial pulses is much more frequently found with the lesion in this position than anywhere else. On percussion a very large area of dulness may be found in the upper sternal region, and on auscultation the double cardiac sound, or a systolic murmur followed by the second sound, or a systolic followed by a diastolic murmur, may be determined. The respiration is often characterised by a loud rough stridor, and on auscultating the chest this may be heard throughout its

most distant regions. When this is the case, there is frequently obstruction to respiration with subjective symptoms of disturbance. Tracheal tugging is almost, if not always, pathognomonic of aneurysm of this portion of the aorta. Aphonia belongs especially to an affection in this position. Interference with swallowing is extremely common, and there are frequent complaints of a feeling as if the food reached a certain point, and was there arrested. Pain is not such a marked feature of aneurysm in this position as when the ascending aorta is the seat of the disease, yet from implication of the cardiac plexus or its connections, severe pain in all respects resembling that of angina pectoris is present. Rupture may take place into the trachea and bronchi, the œsophagus, the great veins, the pleura, or the mediastinum.

Aneurysm of the *descending portion of the arch of the aorta* is somewhat less definite as regards its physical signs than when in the two situations just considered, and in many instances it remains to all intents and purposes latent. When it affects the descending portion of the arch, and the upper portion of the descending aorta, it may give rise to an alteration in the præcordial appearances. The apex beat, for example, may be altered in its position on account of transposition of the heart towards the right, and when that is the case, the significant doubling of the impulse, or "double jogging" of Hope, may be made out on palpation. Percussion of the præcordia determines more accurately the position of the heart, but usually reveals no other area of dulness, and auscultation probably fails to give any results. On percussion of the chest behind, an area of dulness may be made out to the left of the vertebral column, about the level of the third, fourth, fifth, and sixth dorsal vertebræ, and auscultation of this area may reveal distinctive symptoms. Most commonly the stethoscope reveals a single sound—the accentuated arterial sound; but at other times two sounds are present, or instead of any such propagation of the cardiac sounds, there may be a systolic murmur. Sometimes there is some delay in the femoral pulse, but this cannot be regarded as a frequent symptom. Occasionally there is considerable dilatation of the veins of the chest in consequence of pressure upon the azygos veins. Difficulty

in swallowing is extremely common, in fact, this is usually the symptom which calls the attention of the patient to the fact that there is anything wrong. Well-marked wheezing may be heard by the bystander, and on auscultation there is not uncommonly characteristic stridor over the left side of the chest. Sometimes, however, great changes in the physical signs furnished by the left lung may be discovered, such as diminished movement, loss of vocal fremitus, impairment of clearness on percussion, and absence of breath and voice sounds; these may point to occlusion of the left bronchus, and commencing collapse of the lung. In other cases, increase of the vocal fremitus, dulness on percussion, harsh vesicular, or even bronchial or cavernous breathing, with moist accompaniments and foetid breath, may show that gangrene has resulted from pressure on the root of the lung. Pain is sometimes present in the left side of the chest, and may follow the line of the intercostal nerves corresponding to the position of the aneurysm, or it may radiate towards the left shoulder and arm. Cases have been described in which the sac has eroded into the spinal canal, and produced symptoms of irritation or depression of spinal functions. Changes in the pupil of the left eye are common.

Aneurysm of the *descending thoracic aorta*, especially if so situated as to be close to the diaphragm, commonly gives expansile pulsatory appearances, and it may even cause enlargement of the affected side. Sometimes the diffuse pulsation which it produces is accompanied by a thrill, which can easily be determined. Dulness on percussion shows approximately the extent of the aneurysm, and auscultation usually reveals either a single sound or a late systolic murmur. No case has ever afforded me an opportunity of detecting either a double sound or a double murmur, yet it is conceivable, although the aneurysm is situated at a considerable distance from the heart, that a double arterial sound might be present, or in the case of aortic incompetence, a double murmur might be found. There is still some trouble in swallowing when the lesion is in this position, and regurgitation of food is often found. Stridor is never produced by aneurysm close to the diaphragm, but, on account of considerable compression

of the left lung, there may be interference with breathing, shown by dyspnœa, while cough, sometimes with blood stained expectoration, increased vocal fremitus, comparative dullness, harsh, vesicular, or even bronchial breathing, with increased vocal resonance, and sometimes moist accompaniments, may show that some consolidation is produced by compression. When rupture occurs, it usually takes place into the pleural sac or the œsophagus, but it not infrequently opens into the lung, and sometimes into the spinal canal.

Aneurysm of the *abdominal aorta* is in a position readily accessible, and most of its symptoms are therefore easily elicited. A pulsatile swelling may be seen, and the movements thoroughly investigated by palpation, while its size may be ascertained both by palpation and percussion. Auscultation usually furnishes evidence of a systolic murmur, but sometimes there is only a single arterial sound. The femoral arteries not infrequently show a delay of the pulse, as compared with their normal relation to the apex beat and the radial pulse. Dysphagia is usually shown by regurgitation of food immediately after it has been swallowed, while attacks of vomiting may result either from direct interference with the stomach, or from reflex disturbance through some of the splanchnic nerves. The intestinal functions are often modified, and diarrhœa or constipation may result. An icteric tint of the skin is sometimes found when the aneurysm is so situated, and so extensive as to interfere with the liver. The discoloration of the skin forming one of the symptoms of Addison's disease has been observed from interference with the semilunar ganglia or their connections. Pain is usually experienced, and is most commonly of a colicky character radiating throughout the abdomen, but when the aneurysm is situated lower down, it may interfere with lumbar and sacral nerve roots, and thus give rise to painful sensations darting around the lower part of the body, as well as down the inferior extremities. Paralysis of the lower extremities has been described as a result of pressure upon the anterior nerve roots. Aneurysm of the abdominal aorta most commonly ruptures into the retroperitoneal tissues, and spreads in every direction upwards and downwards. Sometimes, however, the escape takes place into the general



peritoneal cavity, while in rare cases, through the formation of adhesions to the abdominal viscera, with subsequent erosion, there may be a discharge of the aneurysmal contents into one or other of them.

The course of aneurysm is almost entirely dependent upon three factors:—the condition of the arterial walls, the integrity of the aortic cusps, and the energy of the cardiac muscle; the general state of nutrition of the body, as a rule, however, exercises very considerable influence over the progress of the affection, and it need hardly be added, that the occupation and environment are of the greatest importance. When the arteries have undergone widespread sclerotic changes, so as to resemble rigid tubes, the oscillations in blood pressure are much greater than when the arteries retain their distensibility and elasticity. It follows from this that in such circumstances there is a much greater tendency to destructive changes than when the pressure is regulated and equalised by the normal properties of the arteries. In a similar way, when the aortic cusps are incompetent, there are much greater fluctuations of pressure than when the valves are competent. Aortic regurgitation therefore prevents the conservative process of coagulation of the blood within the sac, while it tends in the direction of rupture. If the energy of the cardiac muscle is excessive, as is certainly the case in many instances of hypertrophy attendant upon aortic aneurysm and arterial sclerosis, the tendency to rupture is greatly increased. On the other hand, when the cardiac energy threatens to fail, there is always a risk of death from asystole. It is unnecessary to add that the habits and surroundings in any case of aneurysm may be such as to exercise a beneficial or deleterious influence on the course of the disease. According to the different combinations existing amongst these different factors, the progress of the disease may be longer or shorter. In many instances, aneurysm exists for a number of years, and does not in the end directly bring life to a termination, death being caused by some inter-current disease, absolutely unconnected with the aneurysm. In another group of cases, aneurysm indirectly produces a tendency towards death, which ensues at last from asthenia.

In a third class, aneurysm more immediately produces conditions which lead towards a fatal result by cardiac failure. In a considerable proportion of instances, death occurs directly by rupture of the sac. This result, when it occurs after a brief period, is commonly found to be associated with a considerable amount of arterial degeneration, incompetence of the aortic cusps, and hypertrophy of the left ventricle, while it usually occurs during exertion. The exertion need not necessarily be of a very violent kind; a fit of coughing, for example, is often quite sufficient to produce rupture, and its fatal results.

DIAGNOSIS.—The recognition of aneurysm, more especially when situated within the thoracic cavity, is often extremely difficult. The diagnosis must be based upon the presence of some of the symptoms which have been fully detailed above; but as every one of these may be produced by lesions of a totally different nature, there can hardly be said to be any pathognomonic symptom of the disease. The decision in any given case must therefore be largely concerned with questions of probability, and, indeed, the diagnosis is very frequently reached by exclusion. The state of the arterial walls, the condition of the aortic cusps, and the size and energy of the heart are probably of the greatest use.

Mediastinal tumour frequently furnishes a considerable number of symptoms absolutely identical with those produced by aneurysm, and the differentiation of the two diseases is extremely difficult. Every pressure symptom may be present. There may be displacement of the thoracic viscera; a pulsating tumour with an area of dulness closely resembling in position and extent that which might be expected in a case of aneurysm; even a systolic murmur; and yet, from the integrity of the arterial walls, the character of the aortic second sound, and the size and beat of the heart, it may seem to be without doubt a new formation in the mediastinum in contact with the aorta.

Mediastinal abscess is not so likely to give rise to diagnostic error. In most instances of this affection there are general symptoms of pyrexia, and sometimes even unmistakable evidence of septicæmia, while there are few, if any,

direct symptoms, and those from pressure are not so definite as might be expected from an aneurysm.

Cardiac enlargement is commonly mentioned as a possible source of error in diagnosis, but in enlargement of the heart, whether from dilatation, or hypertrophy, or both, there is but one point of maximum intensity of impulse, while in the case of aneurysm there are two spots where the pulsation shows maximum force.

Effusion into the pericardial sac can scarcely be mistaken for aneurysm, seeing that the area of pulsation is single, and its intensity is reduced, while the area of dulness has a characteristic outline, and the sounds on auscultation are diminished in force.

Pulsating empyema seems to be an occasional source of error, yet in such a condition the position of the area of dulness, and the alteration in the physical signs revealed on auscultation, are in themselves sufficient to establish a differential diagnosis.

It is possible that changes in the pulmonary tissues, of tubercular, syphilitic, or cancerous nature, might, if in contact with the heart or aorta, give at once a conducted impulse, and propagated heart sounds. Careful examination of such cases, nevertheless, always shows that the position of the alteration is incompatible with the presence of aneurysm.

PROGNOSIS.—Based, as it is, upon an irremediable change in the tissues of the greatest of the blood vessels, the outlook in aortic aneurysm is always serious, and the prognosis is usually therefore somewhat grave. It must always be dependent upon the facts which dominate the course of the disease in any case under consideration, such as the state of the arterial walls, the aortic valves, and the cardiac muscle, together with the resulting conditions of blood pressure. The degree of general nutrition is also of importance, seeing that it largely controls the circulation. Two other considerations are of great importance in prognosis. Speaking generally, the gravity of the prognosis is directly proportional to the size of the aneurysm; the larger it is, the more liable is it to undergo rupture, in accordance with well-known hydrodynamic principles. The general habits and surroundings

of the patient are most influential either for good or evil. These are simply the conditions which have been above referred to as exercising a powerful influence upon the course of the disease, and the prognosis is necessarily entirely dependent upon them.

TREATMENT. — The subject of treatment, it must be confessed, is somewhat discouraging. In very many cases little can be done except to palliate suffering. In a considerable proportion, however, it is possible, by careful treatment, not only to alleviate the symptoms, but even to aid in bringing about recovery. It must never be forgotten that the results of post-mortem examinations furnish a very considerable number of instances in which aneurysm has been unsuspected during life and has been seen in a condition of more or less complete obliteration after death.

The one great aim in all cases is to favour the coagulation of the blood in the sac. This may be done by diminishing the pressure and velocity of the blood, by the introduction of agents which will directly induce coagulation, or by producing changes in the walls of the sac.

Reduction of the pressure and velocity of the blood may be caused by the simple methods of absolute rest and restricted diet. Rest is imperatively demanded, and, if it be possible, it should be complete. By means of rest alone, without any modification of diet, and without the addition of any drug, there is a considerable change in the number of pulsations and in the pressure of the blood. The blood pressure, further, does not undergo much variation, and the natural tendency to coagulation within the sac is thereby increased. While absolute physical rest is enjoined in cases where it may seem to be necessary, and they form the greater proportion of cases of aneurysm, there should also be, as far as is possible, a cessation of mental effort, since this also is attended by changes in blood pressure and pulse rate. The diet, further, should be, as Douglas Powell put it, "restricted in quantity, but enriched in quality." According to Morgagni, the recumbent posture and starvation diet were employed by Albertini and Valsalva, along with other means, which will be mentioned in the sequel. The combination of



absolute rest and appropriate diet formed the treatment introduced by Tufnell. He recommended an extremely restricted diet:—for breakfast, 2 oz. of bread and butter, and 2 oz. of milk; for dinner, 2 or 3 oz. of meat, along with 3 or 4 oz. of milk; and for supper, 2 oz. of bread and 2 oz. of milk. It is believed that this low diet lessens the amount of blood; it certainly reduces the blood pressure, and, therefore, favours coagulation. It is further held to increase the amount of fibrinogen, and in this way to aid the process of recovery by deposition within the sac. This is, however, doubtful.

Certain drugs are of importance in this connection, inasmuch as they assist rest and diet in the treatment of aneurysm. Nay more, we may say that even in spite of deficient rest and careless diet, some of these remedies are still beneficial.

The most famous of such remedies is iodide of potassium. This drug, first introduced by Graves of Dublin, as a means of alleviating painful affections of the fibrous tissues and nervous system, was observed by Craig to have produced perfect relief from pain in a case of aneurysm, as he stated in a private communication to Balfour. Somewhere about the same time the use of iodide of potassium appears almost simultaneously to have been investigated in Europe and Asia by different observers. Bouillaud in Paris, and Chuckerbutty of Calcutta, found that great relief was obtained by the use of the iodide in cases of aneurysm, and that almost complete recovery ensued if the drug were sufficiently persevered with. Roberts somewhat later employed this remedy in a large number of cases, but it is to Balfour that we owe the extended employment of this drug.

Unfortunately, we do not yet understand the *modus operandi* of iodide of potassium. It was held by Chuckerbutty that the drug increased the coagulability of the blood. This, however, is more than doubtful when we consider that the remedy is one which has such a marked effect as a deobstruent. It is well known that iodide of potassium has some influence in reducing blood pressure and in diminishing the frequency of the heart. The amount of reduction of blood pressure, however, is extremely small, and the diminution in

the rate of the heart is inappreciable with ordinary doses of the drug. It has been shown by Sée and Lapique that the first effect of iodide of potassium in a small dose is to increase the blood pressure, as well as to diminish the frequency of the pulse, and that, if the dose is increased, the blood pressure falls, while the rate of pulsation rises. Balfour strongly recommends that, in the administration of iodide of potassium, such a dose should be administered as will produce some reduction of the blood pressure without any acceleration of the pulse. He holds that the essential process whereby relief is obtained is through thickening and contraction of the wall of the sac. We may admit it to be extremely probable that, under the influence of iodide of potassium, the nutrition of the walls of the sac, as well as of the whole of the arterial system, undergoes improvement, and that some of the adventitious products may be relieved; but that there is any specific influence of this kind may well be doubted. One of the most remarkable effects produced by iodide of potassium is the rapid disappearance of painful sensations. This is, without a doubt, the most constant and, at the same time, one of the most useful results obtained by using the drug. The iodide does not only remove or lessen the continuous dull aching attendant upon aneurysm, but it also obviates or arrests the anginous attacks which occur from time to time. How it does this is still obscure, and we are just as little able to explain the analgesic effect of the drug as to explain the undoubted fact that its administration aids in the relief of the other symptoms of aneurysm. The dose of the drug used to be large—almost as great as that employed in tertiary syphilis—nowadays it is found that results quite as satisfactory in every way may be obtained by the exhibition of doses not exceeding five or ten grains.

Certain other remedies have been employed instead of iodide of potassium. Amongst these may be mentioned the nitrite series, including nitro-glycerin, nitrite of sodium, and nitrous ether, as well as the modern synthetic group of which phenazone may be cited as an example. The effect of these drugs is in every way less valuable than is the case with the iodides.

The internal administration of remedies which are believed to act as astringents has often been recommended. Acetate of lead was vaunted by Dusol and Legroux, while tannin, as well as ergotin, has been very frequently employed. The effects of these drugs cannot be regarded as thoroughly established, but recoveries have been reported.

It is often necessary to add certain auxiliary remedies in order to relieve symptoms. Morphine, for example, must in many cases be employed from time to time as an ancillary remedy, in order to allay pain; and some hypnotic, it may be an opiate, or one of the newer remedies, may be urgently required from time to time.

In addition to the use of drugs, we have certain other means at our disposal whereby the symptoms of aneurysm may be alleviated by reduction of blood pressure. One of the simplest of these methods is general blood-letting, originally proposed by Albertini and Valsalva, strongly advocated by Hughes Bennett, and also recommended by Fagge. The abstraction of four or five ounces of blood from the arm often brings about a striking amelioration of the distressing symptoms by relieving the pressure effects. The influence of this method of treatment may justly be regarded as in every way analogous to that of the drugs which lower the blood pressure by dilating the arterioles. The effects are from the nature of things evanescent, since the blood pressure speedily rises again; the method may nevertheless serve to turn a dangerous corner.

Diminution of pressure within the sac by compression of the vessel on the proximal side, such as is often so serviceable in aneurysm of the peripheral arteries, is inapplicable in the case of the thoracic aorta, but it has been employed with benefit by Murray, Moxon and Durham, Greenhow, Heath, and Philipson in abdominal aneurysm.

Another class of remedies consists of various agents brought to bear directly on the contents of the sac. Amongst these may be mentioned the injection of chemical substances, such as perchloride of iron. The results of these attempts to produce coagulation are not such as to tempt their repetition.

Electrolysis has been greatly employed by Petrequin,

Ciniselli, and Duncan. The method adopted is to pass a continuous current through the contents of the aneurysmal sac. Two needles carefully insulated, except at their points, and connected with the poles of a galvanic battery, are passed into the sac, care being, of course, taken that the points do not touch each other. It is, perhaps, better to employ only one needle connected with the positive pole, and to connect the negative pole with a large rheophore applied to the surface of the body in the neighbourhood. In this way a firm clot is sometimes obtained. Several patients treated by galvanism in this way have been under my care or under my observation. The result in none of these cases was very satisfactory. Probably, however, in every instance the treatment was only adopted when it was obvious that all other means would fail. It seems to me quite likely that this method of practice has scarcely ever had a perfectly fair trial.

Another system of treatment, originally introduced by Moore, but usually associated with the name of Loreta, consists in the introduction of metallic wire into the aneurysmal sac, with the aim of producing coagulation of the blood upon the foreign body so inserted. Fine iron wire is the material which has been most commonly used, but fine wire made of other metals has also been employed, and horse hair has also been introduced. The general effect of this procedure is unfavourable; it is apt to lead to inflammation, so that it aggravates the symptoms and precipitates the termination of the disease. The results of the recorded cases down to 1887—2 recoveries in 16 cases—have been collected by White and Gould. They are certainly not such as to encourage sanguine expectations.

A combination of the methods of Ciniselli and Moore has recently been adopted by Hershey. It consists in the introduction of a coil of gold wire into the sac, and the passage of a continuous current through it. By this means in one case a most successful result was attained.

Macewen has been remarkably successful in the treatment of aneurysm by a method introduced by himself. His procedure is extremely simple, consisting in transfixion of the aneurysm with a needle, and manipulation of it within the



sac. After rendering the skin thoroughly aseptic, the needle is introduced so as to penetrate the sac, and pass through its cavity until it comes into contact with the further side, which it should touch without doing anything more. The needle may then be left, so that the impulse of the blood current may move it about in such a way as to produce irritation of the inner wall of the sac, or it may be moved about by the operator, so as to tear gently the lining of the sac. If the former plan be followed, the needle should only be left a few hours, but Macewen says its retention for twenty-four or thirty-six hours appears to have a greater effect. He is of opinion that it should never remain more than forty-eight hours in the aneurysm. If the other method be employed, after moving the needle backwards and forwards over the opposite wall of the aneurysm for ten minutes, the point should be shifted to another spot without withdrawing it; after having manipulated the new area, another spot may be dealt with, and the process continued in this way, until the greater part of the internal surface opposite has been treated. In Macewen's hands this method of treatment has been markedly successful. No opportunity has yet been allowed me of practising it on any patient under my own care, but some cases, which have been under my observation, induce me to speak in the most favourable way in regard to it.

It is but rarely that the surgical methods of treatment applicable to peripheral aneurysms, such as compression or ligation, can be employed in those of the thoracic or abdominal aorta.

In presenting some cases of aneurysm as examples of common clinical and pathological features, they will be grouped in a definite manner:—a case of fusiform aneurysm in its most usual position; cases of sacculated aneurysm in different situations, to illustrate the facts of localisation; cases to show the relations of aneurysm to the radial pulse; cases of affections which might be mistaken for aneurysm; and a case to show how long life may be maintained and symptoms alleviated by appropriate treatment.

CASE 70. *Dilatation of Ascending Aorta and Innominate Artery.*  
—J. B., aged 54, gardener, frequently consulted me in the out-patient

department of the Royal Infirmary on account of cough and dyspnœa. The family history of the patient was somewhat indefinite, but he had a strong impression that his father had died of heart disease, while it was absolutely certain that one of his brothers had been cut off in early life in consequence of some form of heart affection. His social conditions had



FIG. 199.—Dilatation of ascending aorta in Case 70.

always been satisfactory. The previous health called for but little remark, except that, about ten years before he came under observation, he had suffered from attacks of faintness. The illness for which he sought advice began a few months before he was first seen by me, and he believed that it had been induced by a violent fit of coughing. The general appearance of the patient was healthy. The alimentary system

revealed no symptoms of an abnormal kind. He had no subjective phenomena connected with the circulation.

On inspection, there was a considerable amount of throbbing in the carotid arteries, and this was more especially the case on the right side. The apex beat occupied the fifth intercostal space, 4 in. from mid-sternum; it was somewhat diffuse and sustained. Palpation of the upper region of the chest to the right of the sternum gave rise to a feeling of impact, but it was by no means very distinct. The area of cardiac dulness was found to extend considerably further upwards than the normal—in fact, it reached the level of the episternal notch and the upper margin of the clavicles. Just below the clavicles it extended 3 in. to the right, and 2 in. to the left of the middle line. At the level of the fourth costal cartilage it was 3 in. to the right and 4 in. to the left of that line, and in the fifth intercostal space the border of the heart was 5 in. from mid-sternum. Auscultation revealed a systolic, as well as a diastolic, murmur in the aortic area, and a distinct separate systolic mitral murmur in the mitral area. There was a distinct difference between the two radial arteries, the pulse wave in that of the right being somewhat smaller than that in the left, but there was no retardation of either. There was no evidence of interference with the respiratory system, and the only other symptom of any importance was the presence of some myosis of the right eye.

The diagnosis in this case was fusiform dilatation of the ascending and transverse portions of the aorta. For some time the patient persistently declined to enter the Infirmary as an inmate, but as, in spite of the continuous employment of iodide of potassium and tonic remedies, he steadily lost ground, he at last consented to become an in-patient, and was placed in Ward 22, at that time under my care. Notwithstanding every means to avert it, he rapidly became worse, and manifested evidences of cardiac failure, of which he died suddenly.

The post-mortem examination revealed a considerable dilatation of the ascending and transverse portions of the arch of the aorta. The aortic cusps were thickened and incompetent. The left ventricle was somewhat dilated and greatly hypertrophied. The appearances are seen in Fig. 199.

The case furnishes a good example of simple dilatation of the aorta, with lesions of the cusps from sclerotic changes.

The following instances form a regular series:—

CASE 71. *Aneurysm of the Ascending Part of the Arch of the Aorta.*—

Arthur M., aged 57, formerly a soldier, and latterly a railway porter, was admitted to the Royal Infirmary under my care on account of pain in the chest. His father died from the effects of an accident at the age of 40; his mother of bronchitis when 75; of his brothers and sisters he could tell nothing. He was the father of six children, who had always been in good health. In early life he suffered great hardships both in the Crimean War and the Indian Mutiny, and since his discharge he

underwent a great deal of hard work, more especially in the lifting of heavy weights. His health, however, had been very good until the illness for which he came to the Infirmary. About eighteen months before his admission, he began to suffer from severe pain in the right side of the chest, which extended upwards to the shoulder and down the arm of that



FIG. 200.—Aneurysm of the ascending aorta ; the position of the rupture is denoted by the pencil which is inserted through the opening.

side. He nevertheless continued to do his work, although with increasing difficulty, inasmuch as breathlessness began to trouble him, and gradually became more severe.

The patient when examined was found to have these symptoms to which reference has just been made, and he had also a hard dry cough, but this cough was not of the harsh strident character produced by pressure on the trachea. There was no difficulty either in breathing or in swallowing.



On inspection the right pupil was seen to be larger than the left. The right side of the chest at the level of the second, third, and fourth costal cartilages, with the intercostal spaces between them, was prominent, and distinct pulsation was seen over the area of the swelling. Palpation showed that the impulse was expansile, but not accompanied by any thrill. The apex beat was in the sixth left intercostal space 6 in. from mid-sternum. The cardiac dulness extended outwards to the right 4 in. at the level of the second,  $3\frac{1}{2}$  in. at the third, and 3 in. at the fourth costal cartilage. To the left it extended  $5\frac{1}{2}$  in. at the level of the fifth cartilage. On auscultation, loud systolic and diastolic murmurs were heard over the entire præcordia. The walls of the radial arteries were wonderfully soft and healthy. The pulse was bounding and collapsing, equal both in time and size on each side; it was perfectly regular and its rate was about 80 per minute. There was some comparative dulness of the upper lobe of the right lung, obviously due to the direct effect of pressure on the lung itself. Both lungs elsewhere were free from any sign of disorder. The patient presented no evidence of any disturbance connected with the other systems.

In this case there could be no doubt as to the diagnosis of an aneurysm arising from the right side of the ascending part of the aorta. The patient was kept absolutely at rest and treated by means of iodide of potassium with appropriate diet, and, when the pain rendered it necessary, small doses of morphine were administered. For some time he appeared to undergo a certain amount of improvement, but the symptoms afterwards became more severe, and he died very suddenly one morning, apparently from rupture of the aneurysm. At the autopsy it was found that the heart was greatly hypertrophied, the aortic cusps were thickened and incompetent, and there was a large aneurysm arising from the convexity of the aorta on its ascending portion. The immediate cause of the patient's death was a rupture of the aneurysm into the pericardial sac, where the pericardium surrounds the aorta. The appearances presented by the heart and aneurysm are shown in the accompanying illustration (Fig. 100).

The case furnished a good clinical picture of sacculated aneurysm of the first part of the arch.

CASE 72. *Aneurysm of the Transverse Part of the Arch of the Aorta.*—John R., aged 46, rope-spinner, came under my care in the Royal Infirmary, complaining of pain in the chest. His father, who was 69 years old, was well; his mother died at 51 years of age of some liver affection. He had seven brothers and one sister, all healthy. Of twelve children of his own, six died in babyhood, the remaining children were strong. His surroundings and previous health had been always good. About two years before admission he began to suffer from pain in the left shoulder; this had continued to trouble him ever since, and on the day of his admission his voice suddenly became hoarse and monotonous. Such were his complaints when he was received into the ward.

On examination, it was found that there were no other subjective symptoms—no evidence of such pressure on the trachea, œsophagus, veins, or lungs, as to produce interference with their functions. The præcordia showed no bulging, but there was an evident pulsation in the upper sternal region. The impulse in this neighbourhood was distinctly felt on palpation; it was unaccompanied by any thrill. The præcordial dulness was found to be considerably increased upwards. At the level of the second costal cartilage the dulness extended  $2\frac{1}{2}$  in. to the right, and an equal distance to the left of the mid-sternal line, opposite the third rib the figures were 3 and  $3\frac{1}{2}$ , while at the fourth rib they were  $2\frac{1}{2}$  and  $4\frac{1}{2}$ . No murmur could be heard over the heart, but the second sound was greatly exaggerated over the aortic area and the adjacent parts of the manubrium sterni. The pulse in the right radial artery was much larger than in the left, and it was earlier in time. On elevating the thyroid cartilage, a distinct downward dragging was found with each systolic phase. Laryngoscopic examination of the larynx and trachea revealed, as was anticipated, a complete paralysis of the left vocal cord. There were no further physical signs of disturbance in connection with any of the other systems.

There could be no doubt that in this case there was aneurysm of the transverse part of the aortic arch. Under treatment, by means of iodide of potassium and rest, the subjective symptoms somewhat improved, but there was little change in the physical signs when he left the hospital.

The case exemplifies many of the features of aneurysm of the transverse part of the arch.

*CASE 73. Aneurysm of the Descending Part of the Arch of the Aorta.*—William M., aged 46, engineer, came under my care in the Royal Infirmary for breathlessness, cough, and weakness. He could give no facts in regard to his father, but his mother was alive, and, in spite of being 70 years old, was still a strong woman. He had no brothers or sisters. His surroundings had always been favourable, but his work was hard. He had never suffered from any serious illness. About a month before admission his breathing had become troublesome, and, after having struggled with breathlessness for some time, a cough was developed with some indefinite pain in the back and chest. The patient had no symptoms of disorder of the digestive system, and in particular no difficulty in swallowing.

The apex beat was in the fifth left intercostal space 5 in. from mid-sternum. The cardiac dulness extended from 2 in. to the right to 5 in. to the left of the mid-sternal line, at the level of the fourth cartilages. On auscultation there were murmurs both of systolic and diastolic rhythm referable to the aortic orifice. There was no difference in the radial pulses, which had a character approaching that of Corrigan. On percussion of the chest posteriorly, there was an area of dulness to the left of the spinal column, beginning at the level of the seventh cervical

and extending as far down as the third dorsal spine, a distance of  $3\frac{3}{4}$  in., and over this patch the heart sounds were heard with great distinctness, but no murmur was audible. A deep-toned rhonchus was heard over great part of the left lung posteriorly.

These facts led to the diagnosis of aneurysm of the descending portion of the arch. Rest and iodide of potassium speedily relieved the more important symptoms, but the patient became tired of hospital and left before any conspicuous benefit could be obtained.

In this case it was quite clear that the symptoms and physical signs might have been explained by the diagnosis of an aneurysm arising from the descending part of the arch of the aorta, or of a tumour in the posterior mediastinum, and, if it had not been for the presence of the affection of the aortic orifice, there would have been little evidence in favour of either view as against the other. The fact, however, that there was a lesion of the aortic cusps was a strong point in favour of the diagnosis which was adopted.

CASE 74. *Aneurysm of the Descending Thoracic Aorta.*—John K., aged 45, sea-cook, was admitted under my care in the Royal Infirmary complaining of pain in the chest. His father had died of some convulsive attack, and his mother of phthisis. He never had any brothers, but of two sisters one was in good health, the other had died of typhus fever. His surroundings had been those of a seaman, but he had gone through some severe experiences, one of which must be specially referred to. The patient had suffered from malaria and typhus fever, and about eighteen years before admission he had contracted syphilis. Sixteen years before entering hospital he underwent great hardships. His own narrative showed that when rounding Cape Horn in the barque *Homeward Bound*, with a cargo of coal, and a crew of fourteen hands all told, smoke was discovered coming from the fore-hold on the 19th of June 1878, while the ship was labouring heavily in a high sea. After fighting the flames for five days, it was clear that the barque must be left. When the crew were about to take to the lifeboat, they could not find the captain; he had disappeared during the night. The remaining thirteen had great sufferings, being adrift from the 24th of June until the 10th of July, from which four died. Two Germans jumped overboard in a distraught condition; the mate cut his throat the very night before they were rescued. Only six were left in the boat when it was picked up by the Italian barque *Giranna*, of Genoa, bound for New York, and but three lived to land at New York, which was reached on the 21st of August. It may be added that the second mate and the boatswain, who were, along with the patient, the sole survivors, died some years previously, and he was therefore the last of the crew of the ill-fated barque. Ten months before admission, he began to suffer from pain in the left side of his

chest, but continued to work at the docks for five months, after which he found work out of the question.

On examination it was found that, although the digestive processes were carried out very well, there was considerable difficulty in swallowing.



FIG. 201.—Aneurysm of the descending thoracic aorta.

After the food had passed down a certain distance it appeared to meet with some obstacle, and to pass it only with a painful effort. The chest was observed to pulsate very strongly in the entire lower part of the left side, in front as well as behind. The sensation experienced by the hand



when applied to the thorax was that of an expansile impulse, and it was not accompanied by any thrill. The cardiac dulness was found to extend 2 and  $4\frac{1}{2}$  in. to right and left of mid-sternum at the level of the fourth costal cartilage. Posteriorly there was a patch of dulness extending along the left border of the vertebral column from the sixth to the twelfth spine, measuring  $6\frac{1}{2}$  in. vertically, and 5 in. horizontally. On listening over the præcordia the second sound in the neighbourhood of the aortic cartilage was greatly accentuated, but there was no murmur over any part of the cardiac region except at the xiphoid cartilage, where a murmur was audible half-way between the first and second sounds. Over the patch of dulness posteriorly the two heart sounds were heard with great distinctness, but no murmur could be detected in this position. The radial arteries were almost healthy, the tension was moderate, the rate of the pulse 72, and the rhythm regular.

In this case the heaving pulsation and dull area in the lower part of the left side of the chest, over which the heart sounds could be heard so distinctly, made it almost certain that we had to deal with an aneurysm of the descending aorta; and the accentuation of the second sound in the aortic area rendered this more probable. The dysphagic symptoms made it clear that the sac must be situated above the lower end of the œsophagus, and the diagnosis was that of aneurysm of the descending aorta arising above the pillars of the diaphragm, and extending towards the left.

The patient was treated in the usual way by means of complete rest and iodide of potassium. The pain was so severe and continuous that it was found necessary to administer opiates freely. In spite of the treatment the patient did not make satisfactory progress, and the question of adopting electrolytic treatment was under discussion, when he died suddenly during a fit of coughing.

The post-mortem examination showed an aneurysm of large size in the position suspected, and the immediate cause of death was rupture of the sac into the left pleural cavity. The vertebræ were much eroded. The appearances of the heart and aorta, with the large aneurysm, are shown in the preceding illustration (Fig. 201), taken from the right side. A flexible tube has been passed into the sac at the site of the rupture, and can be seen coming out of the lower cut end of the aorta.

The case illustrates the clinical features of aneurysm of the descending thoracic aorta very thoroughly.

CASE 75. *Aneurysm of the Abdominal Aorta.*—Jane A., aged 43, laundress, was under my care in the Royal Infirmary on account of swelling and pain in the left side. Her father, who died at the age of 60, was a rheumatic man; her mother died of acute pneumonia at 40. All her

brothers and sisters died in infancy except one brother, who was in good health. The patient was married when 16 years old, and had two children—a boy who died when 2 years old, and a daughter now married and the mother of three children. Her surroundings were not unfavourable, but her occupation exposed her to cold, and her food had sometimes been scanty.



FIG. 202.—Aneurysm of the abdominal aorta. The two tubes pass from the aorta into the sac, are continued into the aorta below, and emerge by means of the two iliac arteries.

She never had rheumatism, but suffered once from erysipelas, and the year before her admission she had been in the ward with pain in the chest and breathlessness, accompanied by jaundice and dropsy. Although she lost these symptoms, she never quite recovered, and six months before being admitted for the second time, she began to experience a sharp pain in the left side. The pain, although continuous, was aggravated by

exertion, and was followed, three months later, by a swelling in the left hypochondrium, which gradually increased.

The patient was obviously anæmic, with bloodless lips and gums. She lay on the back and left side, and could not turn on her right side without greatly increasing the pain. Her teeth were bad, and her tongue foul. She suffered from much thirst, and had little appetite. Once or twice after admission there was some hæmatemesis, but no melæna. The area of the stomach was within healthy limits, and the hepatic dullness extended from the fifth rib to the costal margin in the mammillary line, a distance of 6 in. A pulsating tumour was visible in the left hypochondrium; it was expansile on palpation, but could not be fully investigated in this way, as pressure produced great pain. It was dull on percussion, and the dullness merged in that of the spleen, heart, and liver. In the left anterior axillary line it extended from the sixth rib to a point just below the costal margin, and measured 8 in. A loud systolic murmur of soft character was audible over the whole of this dull area.

In addition to pain, the patient suffered from palpitation and



FIG. 203.—Tracing with Marey's stethograph, from Case 75.

dyspnœa. There was much pulsation in the suprasternal and supra-clavicular spaces. The apex beat was diffuse, and occupied the fifth space at the mammillary line. The cardiac dullness was found to extend  $1\frac{1}{2}$  in. to the right and  $4\frac{1}{2}$  to the left of the middle line at the fourth cartilage. In the aortic area there were murmurs of obstruction and incompetence, and there were, further, two independent murmurs significant of tricuspid and mitral regurgitation. The radial vessels were soft, the pulse full, and of moderate pressure; its rate was about 70 and its rhythm regular, but it had to a considerable extent the characters of a Corrigan's pulse. There was no difference between the two radial pulses, and the loss of time between the radial and femoral pulsation was quite imperceptible. There were no symptoms of disorder connected with any of the other organs.

There could be but little room for any difference of opinion in regard to this case save in regard to the exact localisation of the aneurysm, and from the position of the pulsation it seemed to be probable that the aneurysm would be found to have its origin about the celiac axis. Cyrtometer tracings of the two halves of the body at the level of the pulsation showed that the left side was  $2\frac{1}{2}$  in. larger than the right, the figures being respectively  $13\frac{1}{2}$  and 16. Tracings obtained with Marey's stethograph gave in an interesting way the expansion of the whole circumference of the body with each pulsation, as may be seen in Fig. 203.

The patient improved in some measure on iodide treatment, and after a residence of a few weeks in the Infirmary decided upon going home. She had not, however, been there for more than a couple of weeks, when she again presented herself, and was found to be suffering from an attack of a febrile character resembling septicæmia, under which she rapidly succumbed. The autopsy brought to light a large aneurysm of the abdominal aorta at the origin of the cœliac axis, along with obstruction and incompetence of the aortic, and incompetence of the mitral and tricuspid valves. No cause could be found for the septicæmia, the existence of which was amply proved by the presence of purulent deposits in the synovial structures, and in the sheaths of the vessels, as well as by the presence of streptococci.

The appearances presented by the aneurysm are seen in the illustration (Fig. 202).

These five cases form an instructive series, and in order to present their main features in a graphic manner, they are grouped in the following table:—

Symptoms.	Cases.				
	1	2	3	4	5
Tumour . . . . .	×	—	—	—	×
Area of dulness . . . . .	×	×	×	×	×
Expansile pulsation . . . . .	×	×	—	×	×
Murmurs over sac—					
Systolic . . . . .	—	—	—	×	×
Systolic and diastolic . . . . .	×	—	—	—	—
Accentuated aortic second sound . . . . .	—	×	—	×	—
Cardiac murmurs—					
Double aortic . . . . .	×	—	×	—	×
Systolic mitral . . . . .	—	—	—	—	×
Systolic tricuspid . . . . .	—	—	—	—	×
State of radial pulse—					
Right larger and earlier . . . . .	—	×	—	—	—
Pressure on—					
Veins . . . . .	—	—	—	—	—
Nerves—					
Sympathetic . . . . .	×	—	—	—	—
Recurrent laryngeal . . . . .	—	×	—	—	—
Sensory . . . . .	×	×	×	×	×
Trachea—					
Causing “tugging” . . . . .	—	×	—	—	—
Causing stridor . . . . .	—	—	—	—	—
Bronchus—					
Causing rhonchus . . . . .	—	—	×	—	—
Œsophagus . . . . .	—	—	—	×	—
Bones . . . . .	—	—	—	×	—

*N.B.*—In the above table a cross indicates the presence, and a dash the absence of the symptom.



CASE 76. *Aneurysm of the Transverse Portion of the Arch of the Aorta.*  
—J. H., aged 53, wool-packer, came to see me at the Royal Infirmary, 21st June 1895, complaining of hoarseness, and pain in the chest. There were absolutely no tendencies to any particular disease in the family history. His father had been lost at sea; his mother had died of cholera; the entire family had consisted of himself and a brother, who had always been in perfect health. He had been married for twelve years, and had one child, aged 11, who was very well. The social conditions had been excellent. His previous health left nothing to be desired, except in the important particular that he had suffered from

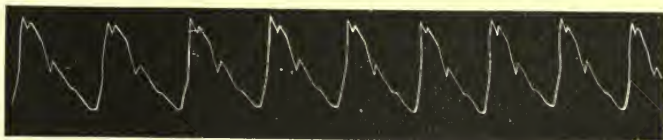


FIG. 204.—Tracing from right radial artery, Case 76.

syphilis thirty years before the date of our interview. The attack for which he sought relief began with hoarseness seven months before his appearance at hospital, which, about a month after its onset, was followed by the development of severe pain behind the sternum; this passed towards the left shoulder and ran down the inside of the left arm to the ulnar fingers. This pain was associated with considerable numbness.

The patient's general appearance was that of health; his height was 5 ft. 8 in., his weight 9 st. 13 lb. He was well-built and muscular. The digestive functions presented no symptoms of special importance.



FIG. 205.—Tracing from left radial artery, Case 76.

Beyond a furred tongue and bad teeth, there were no abnormal features, and, in particular, deglutition was perfect, there being absolutely no trace of obstruction. The glandular apparatus showed no abnormality. The circulatory system gave the subjective phenomena to which reference has been already made. The pain was unattended by any palpitation, but there was distinct dyspnoea on exertion. The cervical veins on the left side were full. There was diffuse heaving of the upper part of the sternum, closely following upon the apex beat, and there was some pulsation in the epigastric region. On applying the hand over the upper part of the sternal region, the pulsation was found to be strong, although diffuse, and with one hand upon this region, and the other over the apex

beat, there was found to be a very slight interval of time, the apex beat slightly preceding the sternal impulse. The apex beat occupied the sixth intercostal space. On percussion it was found that there was great increase in the præcordial dulness, which extended up to the episternal notch, and the upper edge of the clavicles. At the episternal notch the dulness extended  $2\frac{1}{2}$  in. to the right, and  $1\frac{1}{2}$  in. to the left of the mesial plane, and at the level of the fourth costal cartilages its lateral boundaries were  $2\frac{1}{2}$  and 5 in. respectively from mid-sternum. The left cardiac border at the level of the apex beat was  $6\frac{1}{2}$  in. from mid-sternum. On auscultation there were two murmurs, systolic and diastolic, with their maximum intensity in the aortic area, and they showed the mode of propagation characteristic of such murmurs. The arteries throughout the body were rigid, and most of them showed high pressure. There was a distinct difference between the pulsation in the right and left radial arteries, the right being full and quick, while the left was empty and slow. The left also distinctly followed the right in point of time. The pulse rate was 52. The characters of the pulse, as ascertained by the sphygmograph, may be seen in the tracings (Figs. 204 and 205).

The patient's voice was hoarse, but no wheezing was heard. There was a slight cough, dry in character, but not presenting the typical character of tracheal obstruction. There was well-marked tracheal tugging. The examination of the chest produced absolutely negative results, but on examination of the larynx with the laryngoscope, the left vocal cord was found to be absolutely paralysed. There were no symptoms connected with the urinary and integumentary systems, and the only point in regard to the nervous system was that the right pupil was somewhat smaller than the left.

This case furnishes an interesting example of aneurysm of the transverse portion of the arch of the aorta, and it is narrated here in order to show the differences in the radial pulses. It is of interest to observe that there were so few symptoms of pressure upon the hollow viscera passing down the mediastinum, the œsophagus and the trachea being almost exempt from all disturbance, while there was also but little interference with the return of blood from the periphery. Along with the absence of such symptoms, there was considerable disturbance of the nervous tracts by pressure upon different nerves.

CASE 77. *Aneurysm of the Ascending Part of the Arch of the Aorta.*—James T., aged 55, engaged as a porter, complained of palpitation, breathlessness, pain in the right side of the chest, choking sensation after eating, singing in the ears, and weakness of the legs. The patient's father enlisted as a soldier and was lost sight of. His mother died of cholera. He had one brother who died of heart disease, and a sister who was

said to have died of debility. He had eight children, of whom six were in good health, and two had been carried off by whooping cough. He had himself suffered from enteric fever, and had, about twenty years before admission, had some hæmatemesis and melæna. His social conditions had been satisfactory. The illness for which he sought admission began six months before he presented himself, and the earliest symptoms were pain and palpitation on exertion, which gradually increased in severity until his admission.

His lips and gums were blanched ; his teeth were bad, and his tongue furred. When swallowing, food gave him the sensation of being stopped in its descent about the epigastrium, and after it had been swallowed there was considerable throbbing of the heart. The liver dulness extended from the fourth rib to below the costal margin, a distance of  $8\frac{1}{2}$  in. Its inferior border was found in the middle line at the umbilicus. The spleen reached the anterior axillary line. The number of the red blood corpuscles was exactly one million per cubic millimetre, and the hæmoglobin was 25 per cent. On inspection of the thorax, a slight bulging of the right side, beyond the mammillary line, could be detected from the fourth rib to the costal margin. Pulsation was visible in the epigastrium and third right intercostal space. On palpation the apex beat was found in the sixth intercostal space in the left mammillary line. A distinct pulsation could be felt in the second, third, and fourth right intercostal spaces ; this was most distinct 3 in. from mid-sternum, and was perfectly synchronous with the apex beat. On percussion the cardiac dulness was found to extend  $2\frac{1}{2}$  in. to the right and  $5\frac{1}{4}$  to the left of the middle line at the fourth cartilage. There were systolic murmurs, with different characters, and separate points of maximum intensity, in the mitral and tricuspid areas, and over the pulsating arch to the right of the heart. There was a very loud second sound in the aortic area. The radial arteries were atheromatous, the left being especially hard and tortuous ; both had numerous rough deposits, probably calcareous. The left radial pulse was very much smaller than the right. There was a loud systolic murmur over the left clavicle and subclavian artery.

In this case the diagnosis was aneurysm of the ascending part of the arch of the aorta, and the difference in the radial pulses was considered to be caused by an atheromatous change in the left subclavian artery. It was further concluded that the patient was suffering from pernicious anæmia.

As time went on, in spite of arsenic, iron, and iodide of potassium, the patient's condition became worse, the blood assumed more characteristic changes, and he gradually sank.

At the post-mortem examination a large sacculated aneurysm of the ascending aorta was found ; the heart was fatty, its cavities dilated, the mitral and tricuspid orifices enlarged, the aortic and pulmonary cùsps healthy. The left subclavian artery was nearly obliterated by a large patch of atheroma. The liver gave a characteristic reaction with hydrochloric acid and ferrocyanide of potassium.



In this case, therefore, the difference in the radial pulses was quite unconnected with the aneurysm; it may be said, however, that the alteration in the arterial pulses and the development of the aneurysm were due to the same pathological process.

CASE 78. *Aneurysm of the Transverse Part of the Arch of the Aorta.*—Anthony K., aged 41, coal-porter, was admitted suffering from pain in the chest and loss of voice. His father had died in early life of cholera; his mother at the age of 70 of some acute disease, probably pneumonia; his only brother was killed in the Indian Mutiny; one sister was alive and in excellent health, the other sister had died some years before in consequence of her husband's ill-usage. The patient's social surroundings had always been fairly good. His previous health had been excellent. Two years before admission he began to suffer from pains in the chest, general weakness followed, and latterly he had lost his voice.

The alimentary system showed no symptoms except at times difficulty in swallowing. The hepatic dulness in the mammillary line extended to  $5\frac{1}{2}$  in. The spleen, glands, and blood presented no abnormal symptoms. No alteration in form could be seen on examining the thorax, and there was no pulsation visible except the apex beat in the fifth interspace. Palpation revealed nothing save a slight systolic heaving about the manubrium sterni. On percussion the cardiac dulness at the level of the fourth rib was found to extend 2 in. to the right and  $3\frac{1}{2}$  in. to the left of the middle line; it reached a higher level than usual, and in the first interspaces was  $3\frac{1}{2}$  in. across. Loud aortic murmurs, systolic and diastolic, were heard. The radial arteries were almost healthy, with soft elastic walls. The volume was moderate, the pressure low, and the type of pulsation bounding—in fact, it was a Corrigan's pulse. The pulsation in the two radial arteries was absolutely equal. A well-marked capillary pulse could be seen, causing a blush on the forehead. Tugging of the trachea was present, and a characteristic stridor, with harsh brassy cough, from pressure on the trachea. The left vocal cord was seen on laryngoscopic examination to be absolutely motionless from pressure on the left recurrent. The right pupil was widely dilated from pressure on the sympathetic.

This case was an excellent instance of aneurysm of the transverse part of the arch of the aorta, without any alteration in the radial pulse. Under appropriate treatment the patient improved considerably, and was discharged relieved.

CASE 79. *Malignant Stricture of the Œsophagus simulating Aneurysm.*—L. E., aged 43, was admitted to the Royal Infirmary under my care, suffering from loss of voice and difficulty in swallowing. The family history was negative, and the general habits and social conditions were excellent. The patient's previous health had always been good. The



attack for which he came under observation began a month before his admission with some difficulty in swallowing, especially of fluids. It day by day became worse, and led to regurgitation of part of the food, which appeared to be arrested at a point behind the manubrium of the sternum. A little later, pain between the scapulæ began to show itself, and this was followed by the loss of voice. The patient had an excellent appetite, but found considerable difficulty in swallowing all substances, but more especially fluids. Sometimes after eating there was some discomfort in the upper abdominal region. Inspection furnished no abnormal phenomena connected with the circulation. Palpation showed that the apex beat occupied the fifth intercostal space 3 in. from mid-sternum; its characters were in no way modified. On passing the finger deeply into the suprasternal notch, a slight pulsation could be felt, more particularly towards the left side. The cardiac outline was found to be quite within ordinary limits, extending upwards to the upper border of the third rib, and at the level of the fourth rib reaching 2 in. and  $3\frac{1}{2}$  in. respectively to right and left of mid-sternum. On auscultation no symptom of disturbance could be elicited save accentuation of the aortic second sound. The arteries through the whole body were extremely atheromatous, and the radial showed exactly similar appearances on both sides. The vessels were somewhat rigid and tortuous, and the blood pressure was rather above the ordinary standard. The pulsation was perfectly regular and equal.

There was no interference with respiration, and most careful examination of the chest failed to show any abnormality, but on examining the larynx with the laryngoscope, the left vocal cord was found to be paralysed. There were no symptoms connected with any of the other organs.

This case presented some difficulty in diagnosis. The difficulty in swallowing, and the paralysis of the left vocal cord, raised the suspicion that an aneurysm of the transverse portion of the arch of the aorta might be present, and the atheromatous condition of the arteries, as well as the accentuation of the aortic second sound, gave a certain amount of plausibility to such a conception. As there was, however, no abnormal pulsation in any part of the chest, and no increase in the cardiac dulness, it seemed highly improbable that aneurysm could be the cause of the symptoms. Taken in connection with the somewhat rapid emaciation, it seemed to be clearly a case of invasion of the œsophagus by means of some new formation.

After he had been under observation for about a fortnight, in addition to losing flesh rapidly, the patient developed two new symptoms. One of these was that on coughing a

little blood stained mucus was expectorated; the other was the gradual appearance of a harsh brassy cough, and a persistent growling stridor. On gently passing an œsophageal tube, it was found to meet with considerable obstruction. Professor Annandale kindly saw the patient along with me, and by means of the bougie determined that there was an annular stricture of the œsophagus situated just behind the top of the sternum. He therefore concurred in the diagnosis of malignant disease, which was afterwards verified.

CASE 80. *Mediastinal Tumour, yielding Symptoms resembling those of Aneurysm.*—T. A., aged 39, wood-turner, was under my care in the Royal Infirmary on account of hoarseness and dysphagia. The family

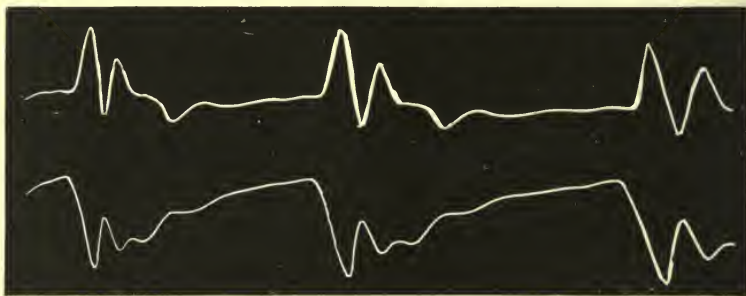


FIG. 206.—Tracing of pulsating areas in Case 80. The upper curve is from the pulsation at the base of the heart; the lower from near the apex;

tendencies were somewhat significant, since his father had died at the age of 52 from phthisis, and his mother at 45 from malignant disease of the liver. His brothers and sisters were in good health, and his four children were perfectly well. His social conditions had always been excellent. As regards his previous health, he had met with an accident to the second right costal cartilage at the age of 27, and, two years before admission, he had suffered from acute pneumonia. Shortly after this attack he began to suffer from throat symptoms, more especially from pain on using the throat in any way. This condition became gradually worse, and was complicated by the development of persistent cough and constant breathlessness, so much so that he found it impossible to keep the recumbent posture.

The patient was extremely emaciated, and weighed 6 st. 13 lb., whereas, two years previous to admission, his weight had been 10 st. 2 lb. His height was 5 ft. 7 in. He was extremely sallow, accompanied by a jaundice tinge. There was considerable trouble in swallowing. The mesenteric glands were enlarged. The liver extended from the upper

border of the fifth costal cartilage to the margin of the ribs—a distance of 5 in. The spleen was somewhat enlarged, and in the inguinal region there was increased size and matting together of the lymphatic glands. On inspection of the præcordia, the apex beat was seen in the fifth intercostal space, and there was, in addition, a distinct pulsation in the second, third, and fourth left intercostal spaces. Simultaneous tracings from the basal impulse and a spot near the apex are shown in Fig. 206. On placing the hand over the præcordia, the apex beat was determined to be 3 in. from mid-sternum; it presented no characters other than those normally found, but over the pulsating region at the base there was a distinct systolic thrill. On percussion, the heart was found to reach the second left intercostal space, and to extend at the level of the fourth costal cartilage 2 in. to the right, and 4 in. to the left of the mesial plane. On auscultation a systolic murmur was detected at the base of the heart, which had its maximum intensity over the centre of the pulsating area in the third intercostal space. The arteries were everywhere soft and compressible, and the blood pressure was below the normal. The left radial artery had a smaller blood wave than the right, but they were absolutely synchronous.

There was a considerable degree of huskiness of the voice. Examination of the chest showed evidences of slight consolidation of both apices, and inspection of the interior of the larynx showed that the left vocal cord was somewhat swollen, but freely movable, while the right was fixed in the cadaveric position. The other systems of the body showed no abnormal symptoms.

There could be little doubt in this case, in spite of a slight superficial resemblance between its symptoms and those which might be produced by aneurysm of the transverse portion of the arch of the aorta, that the lesion was an invasion of the mediastinal glands, probably by tubercular processes. The symptoms of interference with the larynx became rapidly worse, so much so that, to prolong life, it became necessary to have recourse to tracheotomy, which was performed by Professor Annandale. This gave considerable relief, but the patient became extremely anxious to reach home, and he was therefore allowed to leave the Infirmary. His doctor informed me afterwards that he had died on 26th February, but that no post-mortem examination was allowed.

These two cases furnish instructive examples of affections presenting so many features of aneurysm as to give rise to a certain amount of debate before arriving at a definite diagnosis.

CASE 81. *Results of long-continued Treatment of Aortic Aneurysm.*—W. D., æt. 39, now a lamplighter, formerly a seaman, came under my notice during the year 1880 at the New Town Dispensary, complaining of pain in the chest, throbbing, and breathlessness on exertion. His family history was good in every respect. From boyhood he had followed the sea, at first for several years in Her Majesty's Navy, and afterwards in the Merchant Service. During the two or three years previous to coming under my observation he had been engaged as a lamplighter, and, in accordance with the antiquated custom then in vogue in this city, he required to run up a ladder in order to light each street-lamp. In both occupations he had, therefore, undergone a very considerable amount of physical exertion. Many years before he had suffered from specific infection, but, with this exception, his health had always been satisfactory. He was not able to fix very definitely when the symptoms began to trouble him, but they had been present for some months. His symptoms began with pain, but the palpitation and breathlessness developed rapidly after the onset of the pain.

On examining the patient, it was found that there was a distinct bulging in the third and fourth intercostal spaces on the right side, extending outwards to about 3 in. from mid-sternum. This swelling showed well-marked pulsation, and, on applying the hand over the part, the pulsation was found to be of an expansile character. The apex beat was in the fifth left intercostal space,  $3\frac{1}{2}$  in. from mid-sternum. There was no thrill over any part of the præcordia. The cardiac dullness was found to begin at the upper border of the third left costal cartilage. The left border was situated almost 4 in. from mid-sternum, and, opposite the fourth right costal cartilage, dullness existed outwards also to very nearly 4 in. from the mid-sternum line. The cardiac sounds were everywhere normal, with the exception that in the aortic area the second sound was considerably accentuated. Over the swelling there was a soft systolic murmur, which was conducted over the upper part of the chest on the right side, and was also carried into the carotid and subclavian arteries. The walls of the radial arteries were healthy, the vessels being neither rigid nor tortuous. The blood pressure was moderately high, the pulsation was regular, and there was no difference in volume or time between the two radial arteries. There was no interference with any other system of the body; there was no cough or dyspnoea, no interference with the nervous system, apart from the pain from which the patient suffered, and no disturbance of the digestive apparatus.

In this case there were one or two points which rendered the prognosis less grave than it might otherwise have been. The patient was under middle age, the aneurysm arose from the ascending portion of the aorta, and there was no incompetence of the aortic valves. These facts formed the elements of a better prognosis than is often the case.



His condition was fully explained to him, and he was strongly advised to enter the Royal Infirmary, in order to have the best possible opportunity of obtaining relief. To this advice he gladly consented, and he was, therefore, placed under the care of Dr. Brakenridge in Ward 31, on 30th September 1880, where he was treated by means of absolute rest, restricted diet, and iodide of potassium. Under this treatment he rapidly improved, and in about a month he was discharged



FIG. 207.—Heart and aneurysm seen from the right side.

at his own request from the Royal Infirmary, and placed himself once more under my care as an out-patient of the New Town Dispensary. In spite of strong advice to the contrary, he persisted in following his occupation as a lamplighter, and, in consequence of bronchial complication, no doubt due to over-exertion and undue exposure, his symptoms became aggravated, and he was again advised to enter the Royal Infirmary, which he did on the 19th January 1881. He was placed under the care of Dr. Balfour in Ward 32. On this occasion the line of treatment adopted was practically identical with that pursued in Dr. Brakenridge's

Ward, but he remained between two and three months in the Infirmary, and left it very considerably improved.

For several years he was, from time to time, seen by me, and he really acquired considerable skill in the management of his own case. It is perfectly true that he still pursued a vocation involving a considerable expenditure of physical energy, inasmuch as, although he ceased to act as a lamplighter, he undertook the duties of keeper of one of the Edinburgh monuments, which, being a place of popular resort, involved his



FIG. 208.—Heart and aneurysm seen from the left side.

ascending frequently in the course of the day to a considerable height. Such exertion, however, was confined to a short period of the year, and, during the remaining months, he was able to take good care of himself. From time to time he took iodide of potassium, but, unless he had some respiratory or digestive complication, he required no other treatment.

In May 1891 he came under the care of Dr. Craufurd Dunlop. He was then suffering from cardiac failure, for which he was treated with digitalis, and on which treatment he rapidly improved, so as to be able to resume his duties within ten days.

He was again attended by Dr. Dunlop in May 1892, for an attack of cardiac failure, and, although he recovered from this attack, he was never so well afterwards, and died somewhat suddenly on 21st October 1892.

The post-mortem examination was performed by Dr. Bruce, when an aneurysm was found arising from the convex aspect of the ascending portion of the arch of the aorta. The heart was somewhat dilated on both sides, but all the valves were competent. There was no sclerosis of the coronary arteries, and no occlusion of their mouths.

This case forms a most interesting example of a patient who, in spite of somewhat adverse circumstances connected with his occupation, lived a considerable number of years, and finally did not die from rupture of the sac, but from the indirect effects produced upon the heart.

The appearances presented by the heart and aneurysm are well brought out in Figs. 207 and 208. Both show the projection of the sac through the chest wall, and in Fig. 208 the interior of the aneurysm may be seen through the incision made into it.

## RUPTURE OF THE AORTA.

Spontaneous rupture of the aorta in the absence of aneurysm is far from common, yet it has been known since the investigations of the early anatomists and pathologists. The occurrence can scarcely take place save as a consequence of degenerative changes in the arterial walls, but it is extremely difficult to explain why rupture should result instead of aneurysm, except on the supposition of an inherent weakness of the tissues. The following case furnishes an interesting example of the occurrence:—

CASE 81. *Rupture of the Ascending Aorta.*—W. H., aged 16, engaged in a quarry, was admitted to the Deaconess Hospital, 7th January 1897, suffering from breathlessness.

But little could be ascertained in regard to the family history of the patient, since both his parents had died in early life, when he was only an infant, and he had been cared for by some distant relations who knew scarcely any facts about his father and mother. He had throughout his short life been rather delicate, and had latterly been entirely unable to perform the not very onerous duties imposed on him; he had never suffered from any affection likely to damage his heart.

The patient's appearance was, for his years, most remarkable. Tall

—measuring in fact 6 feet 5½ inches—yet stooping to such an extent as to render his stature apparently much less than it was, round-shouldered and exceedingly thin—his weight being only 8 stone 8 lbs.—he presented the aspect of a middle-aged man prematurely old, rather than that of



FIG. 209—Rupture of the aorta in its ascending portion.

a boy. His expression was listless, even apathetic, and his shrunken features were cyanotic in tint. The digestive system showed no departure from a healthy standard, and all the abdominal viscera were of usual size, including the spleen. The blood was, unfortunately, not examined. The pulse during the early days of his residence in the hospital



varied in rate between 80 and 104. The artery was somewhat more rigid than it should have been for the age of the patient, but showed no sinuosity; the rhythm of the pulse was rather irregular and unequal; the individual beats were somewhat tardy yet not sustained. The chest was markedly pigeon-breasted, but showed some prominence of the left portion of the præcordia. The apex beat was in the sixth left intercostal space, nearly four inches from midsternum, and was short and slapping in character. The area of cardiac dulness extended upwards to the third left costal cartilage, and reached from 2 inches to the right to 4 inches to the left of the mesial plane. An aortic systolic murmur was propagated up the cervical arteries, and there were systolic murmurs in the mitral and tricuspid areas. The respirations were, as a rule, somewhat hurried, numbering about thirty-five, but the rate sometimes fell to twenty per minute. There was some cough, with slight frothy expectoration. Physical examination of the respiratory system gave no evidence of disturbance save crepitations at both bases. The urinary, cutaneous, and nervous systems were free from any abnormal symptoms.

The case was one of some difficulty. The aortic systolic murmur might have been produced by some lesion of the cusps, but there was no history of any disease likely to have caused any disturbance of the kind. Taken with the unyielding character of the arteries, it seemed probable that in spite of the youthful age of the patient there was some degenerative process at work, giving rise to sclerosis of the arterial system with aortic dilatation and consecutive cardiac dilatation.

He was kept absolutely at rest, and treated by means of ten-grain doses of iodide of potassium in half ounces of infusion of digitalis. For a few days he appeared to improve, but on 23rd January, when in the act of taking dinner, he suddenly fell back and passed away without a sound. His death was deemed to be due to asystole, but, at the post-mortem examination, which was performed by Dr. Cattnach on the following day, the pericardium was found to be distended with blood, which had escaped from a small linear opening towards the posterior part of the ascending aorta at the highest level of the pericardium. The ventricles were considerably dilated, the mitral and tricuspid orifices enlarged, the aortic cusps healthy, the aorta atheromatous and dilated. The lungs were œdematous. Fig. 209 shows the appearance of the unopened heart, and the position of the rupture, through which a pen has been introduced

The facts present an excellent instance of arterial degeneration and its consequences in early youth, and bring forward with dramatic vividness one of the less common terminations in such conditions.



## APPENDIX I.

### APPLICATION OF RADIOGRAPHY.

SINCE the introduction of the Röntgen rays as practical aids to medicine and surgery, many attempts have been made to render



FIG. 210.—Skiagram of the chest in complete transposition of the viscera.

them useful in the diagnosis of cardiac affections. Many difficulties have stood in the way of realising such aims. Not only

are there serious mechanical obstacles in the way of carrying out the processes, but on the part of the heart itself there are certain impediments. On account of its rhythmic movements it is quite impossible to obtain an absolutely sharp outline of its margins, and considerable scepticism is permissible in regard to the observations, based on radiography, intended to show diminution of the size of the heart as the result of certain methods of treatment.

There are two directions in which the employment of the rays may be of real utility. They may demonstrate a change in the position of the heart, and they may reveal the presence of an aneurysm or intra-thoracic tumour. A beautiful example of their application in cases of the former description is shown in Fig. 210, which is a skiagram of the chest in the case of complete transposition of the viscera, which has already been mentioned and figured on p. 127. It should have been placed in that position, but was not obtained until the sheet had passed through the press. For it my warm thanks are due to Dr. John Macintyre, of Glasgow, whose work on this subject is so well known. It brings out with remarkable clearness the whole bony framework of the chest and upper extremities, and shows the apex of the heart pointing towards the right, while the dense mass of the liver can be made out on the left side.

Dr. Macintyre has been able in several cases to obtain most excellent results in cases of aneurysm and tumour within the thorax.

It may be said by some that radiography is unnecessary in thoracic disease, since the facts which it discloses can be ascertained by other and older methods. It must be conceded that this argument is to some extent true, yet it may well be claimed that every new mode of investigation furnishes at least the possibility of additional knowledge, while the method now under discussion is actually able to reveal an obscure aneurysm or tumour, which would else elude detection.



## APPENDIX II.

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## CHAPTER XV.

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